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RESPIRATORY ADAPTATION AND PULMONARY FUNCTION PARAMETERS IN COMPETITIVE SWIMMERS: A SYSTEMATIC REVIEW OF PHYSIOLOGICAL MECHANISMS AND ENVIRONMENTAL FACTORS

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ABSTRACT

Research Objectives: Competitive swimming uniquely impacts the respiratory system, inducing profound physiological adaptations and environmental risks. This systematic review synthesizes current evidence on pulmonary function, adaptation mechanisms, and airway dysfunction etiology in swimmers.
Methods: A systematic search was conducted up to February 2026. Peer-reviewed studies assessing objective lung function, respiratory mechanics, and environmental exposures in aquatic athletes were included. Data underwent narrative synthesis due to methodological heterogeneity.
Key Findings: The aquatic environment acts as a potent respiratory stimulus. Elite swimmers consistently exhibit supranormal lung volumes (FVC, TLC) and diffusion capacity (DLCO), driven by isotropic growth and alveolar hyperplasia. However, this is counterbalanced by the "swimmer's paradox". Chronic inhalation of disinfection by-products (e.g., trichloramine) combined with massive ventilatory demands induces epithelial damage and oxidative stress, resulting in a high prevalence of exercise-induced bronchoconstriction (EIB) and non-allergic airway hyperresponsiveness.
Conclusions: The impact of swimming on respiratory health appears to be dose-dependent. While structured training is associated with enhanced pulmonary development, high-intensity exposure in chlorinated environments may increase the risk of adverse respiratory effects. Protecting athletes requires sport-specific diagnostic strategies and optimized ventilation systems in aquatic facilities.

KEYWORDS

Competitive Swimming, Pulmonary Function, EIB, Trichloramine, Physiological Adaptation

CITATION

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Introduction

The aquatic environment is a unique setting that imposes exceptional physiological demands on the human body, to which it is not naturally adapted (Pendergast et al., 2015). Swimming is a form of aerobic exercise that requires substantial oxygen input and provides significant benefits to the respiratory system. Movements such as pushing and kicking against water resistance stimulate blood flow to the heart, blood vessels and lungs, forcing almost all muscles to overcome the density of the water. Due to the increased oxygen demand and ventilation rates, regular swimming training serves as an effective method for enhancing lung vital capacity, which is a crucial indicator of physical health and pulmonary function (Hadiansyah et al., 2022).

Body immersion in water, particularly head-out water immersion, triggers profound cardiorespiratory and hemodynamic responses. The hydrostatic pressure of the water compresses peripheral tissues, shifting blood volume from the extremities into the thorax, which significantly increases intrathoracic blood volume. This phenomenon subsequently leads to increased end-diastolic volume, stroke volume and cardiac output. Furthermore, hydrostatic pressure places an elastic load on the chest wall, promoting negative pressure breathing. This external resistance substantially increases the work of breathing and the energy cost for the respiratory muscles (Pendergast et al., 2015).

Given the increased strain on the respiratory system during swimming, the comprehensive assessment and training of respiratory muscles are critical for optimizing athletic performance and minimizing the risk of respiratory-related issues. The strength and endurance of respiratory muscles are increasingly recognized as vital components of overall physical capacity, facilitating efficient oxygen delivery during intensive exercise. Well-trained respiratory muscles allow athletes to sustain higher exercise intensities for prolonged periods and accelerate post-exercise recovery. While traditional spirometry remains a fundamental tool in sports medicine, dynamic evaluation of respiratory muscle strength, such as the S-Index test, are gaining prominence in the comprehensive assessment of well-trained athletes (Kowalski et al., 2025).

However, despite the profound cardiovascular and respiratory benefits of swimming, the specific pool environment exposes athletes to significant health challenges. Prolonged exposure to high ventilation rates in environments full of disinfection products, such as chloramines, has been linked to epithelial damage (Bernard et al., 2003; Font-Ribera et al., 2019), airway hyperresponsiveness and a high prevalence of exercise-induced bronchoconstriction, also called 'swimmer's asthma' (Leahy et al., 2019; Lima et al., 2024; Sepioło et al., 2025). Therefore, evaluating respiratory adaptation in swimmers requires a balanced approach that considers physiological enhancements as well as clinical risks associated with the aquatic environment.

Understanding the interplay between the physical properties of the aquatic environment, cardiorespiratory adaptations and respiratory muscle mechanics is essential in sports science. Therefore, the aim of this review is to comprehensively synthesize the existing literature on pulmonary function parameters and respiratory adaptations in competitive swimmers.

Methodology

Study Design

This study was conducted as a systematic review aiming to synthesize existing evidence regarding pulmonary function adaptations and respiratory mechanics in competitive swimmers, with particular emphasis on physiological mechanisms and environmental factors. The protocol focused on identifying peer-reviewed studies that provided quantitative data on lung volumes and respiratory performance. The review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

Search Strategy and Information Sources

A systematic search was conducted across three major electronic databases: PubMed/MEDLINE, Scopus, and Web of Science. The search encompassed articles published from database inception up to February 2026.

The search strings utilized a combination of Medical Subject Headings (MeSH) terms and free-text keywords:

Population: "competitive swimmers", "elite swimmers", "aquatic athletes", "professional swimmers".

Outcomes: "lung capacity", "pulmonary function", "spirometry", "vital capacity", "forced expiratory volume", "total lung capacity", "respiratory muscle strength", "lung volumes", "exercise-induced bronchoconstriction", "EIB", "asthma".

Mechanisms and environmental factors: "hydrostatic pressure", "chlorine exposure", "disinfection by-products", "trichloramine", "physiological adaptation", "airway hyperresponsiveness", "respiratory muscle training".

Boolean operators (AND, OR) were applied to refine the results. Additionally, reference lists of included studies were manually screened to identify further relevant articles.

Inclusion and Exclusion Criteria

To ensure the selection of highly relevant literature, specific eligibility criteria were established based on the PICOS (Population, Intervention/Exposure, Comparison, Outcomes, Study design) framework:

Inclusion Criteria

Studies were included if they met the following criteria:

1. Peer-reviewed original research articles, observational studies (including cross-sectional and cohort studies), and longitudinal trials.
2. Studies involving human subjects (both male and female and both adolescent and adult) engaged in competitive or high-level recreational swimming.
3. Papers providing objective measurements of lung function (e.g., spirometry, lung volumes, diffusion capacity).
4. Papers that investigated physiological adaptations and/or environmental exposures related to swimming.
5. Studies published in English or providing professional English translations.

Exclusion Criteria

Studies were excluded if they met the following criteria:

1. Case reports, editorials and conference abstracts without full-text availability.
2. Studies involving only animal models.
3. Studies where swimming was only a minor component of a multi-sport intervention without separate data for swimmers.

4. Research focusing exclusively on pathological conditions unrelated to swimming occupational or environmental hazards.
5. Papers focused exclusively on acute exercise responses without baseline pulmonary assessment.
6. Studies that did not report objective pulmonary function measurements.

Data Extraction and Quality Assessment

Data were extracted independently using a standardized data collection form. The following information was recorded from each included study:

- Authors and Year of Publication
- Study Population and Participant Characteristics (including age, sex, and anthropometric data)
- Training Level and Duration
- Pulmonary Function Parameters (e.g., static and dynamic volumes, diffusion capacity)
- Methodology and Diagnostic Tools
- Key Findings.

To evaluate the risk of bias, studies were categorized as high, moderate, or low quality based on selection, comparability, and outcome assessment criteria. This was conducted utilizing standardized critical appraisal tools appropriate for the study design (e.g., the Newcastle-Ottawa Scale for observational studies and the Cochrane Risk of Bias tool for controlled trials).

Data Synthesis and Analysis

Due to the expected methodological heterogeneity in study design, participant characteristics, and outcome measures across the included literature, a narrative synthesis was performed. Studies were grouped according to:

1. Pulmonary function outcomes and respiratory mechanics.
2. Age group (adolescents vs. adults) to account for developmental stages.
3. Training level (elite vs. sub-elite and recreational).
4. Environmental exposure (chlorinated vs. non-chlorinated environments and related airway dysfunctions).

Where sufficient homogeneity was identified, thematic comparisons were qualitatively summarized. Special emphasis was placed on distinguishing between potential training-induced adaptations and pre-existing anthropometric characteristics.

Sources of bias and methodological limitations within the primary studies were critically evaluated and discussed.

This structured approach allowed for a comprehensive and multidimensional narrative synthesis of the respiratory profile of swimmers.

Results

Lung Capacity in Swimmers

The literature consistently highlights the profound positive impact of swimming on pulmonary function, demonstrating significant enhancements in key spirometric parameters across various age groups, training levels and durations. Unlike land-based sports, swimming inherently demands that the respiratory system overcomes the hydrostatic pressure of the aquatic environment. These factors drive profound external and functional adaptations within the respiratory system (Basavaraj et al., 2014).

Short-term Adaptations in Beginners

Evidence suggests that even relatively short periods of swimming training can induce measurable improvements in lung capacity. Gorai et al. (2019) evaluated healthy young adult beginners and observed significant increases in Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV1), Peak Expiratory Flow Rate (PEFR), and Maximum Voluntary Ventilation (MVV) after three and six months of regular swimming sessions. The dramatic improvement in MVV suggests that even a brief period of regular swimming effectively increases respiratory muscle mass and endurance, enabling the respiratory pump to sustain higher ventilation rates without premature fatigue. Similarly, Nicoară et al. (2024) reported that an 8-week program combining swimming and specific apnea exercises resulted in substantial improvements in FVC, FEV1, and Peak Expiratory Flow (PEF) among university students compared to a non-exercising control group, emphasizing the rapid responsiveness of the respiratory system to aquatic stimuli.

These primary observations are strongly supported by a comprehensive systematic review and meta-analysis conducted by Lahart and Metsios (2018). Their analysis confirmed that even in non-elite and non-competitive populations, swimming interventions result in statistically and clinically significant improvements in maximal oxygen uptake and peak expiratory flow, solidifying swimming as a highly potent stimulus for respiratory enhancement across diverse demographics.

Long-term Adaptations and Competitive Swimmers

In highly trained and competitive athletes, pulmonary adaptations become significantly more pronounced. A descriptive study by Basavaraj et al. (2014) involving young male swimmers found that athletes possessed markedly higher lung volumes than non-swimmers. The authors attributed this to the fact that the respiratory muscles, particularly the diaphragm, are forced to generate greater pressure during the respiratory cycle due to water immersion, leading to functional improvements and alterations in the elasticity of the lung and chest wall. The magnitude of these adaptations is striking. For instance, García et al. (2021) demonstrated that elite aquatic athletes exhibit extreme functional expansion, with male and female demonstrating Total Lung Capacity (TLC) reaching up to 133% and 116% of their predicted reference values, respectively. Furthermore, this adaptation uniquely includes a massive expansion of the Residual Volume (RV), which frequently exceeds 135-140% of standard references, a phenomenon rarely seen in land-based athletes.

The developmental impact of intensive training is particularly evident when initiated in youth. Courteix et al. (1997) demonstrated that prepubertal girls undergoing vigorous swimming training (12 hours per week) for one year exhibited significantly larger Vital Capacity (VC), Total Lung Capacity (TLC), and Functional Residual Capacity (FRC) than a control group engaged in moderate terrestrial sports. Beyond merely increasing static volumes, swimming training during prepuberty significantly alters the proportional development of the respiratory system. Courteix et al. (1997) observed that intensive swimming in young girls promoted 'isotropic lung growth'. This means that the alveolar spaces and the airway system develop harmoniously at a similar rate, leading to improved conductive properties of both large and small airways, as evidenced by significant increases in maximal expiratory flows (MEF25-75). This isotropic development during childhood may provide a crucial foundation for the superior dynamic lung function observed in adult swimmers. Addressing the ongoing debate regarding whether the large lungs of swimmers result from intensive training or genetic endowment. Bovard et al. (2018) confirmed that competitive swimmers possess significantly greater lung volumes compared to controls. However, their finding indicated that while swimmers possess significantly larger lungs initially, a season of competitive training during puberty did not further accentuate this enhanced lung size, suggesting that the exceptionally large lungs of competitive swimmers may be an inherent trait rather than solely the result of training adaptation. To further contextualize these findings, the recent review by Rochat et al. (2022) emphasizes that while genetic predispositions and inherited pulmonary traits cannot be excluded, they do not fully explain the phenomenon. The current consensus suggests that intense swim training modifies the actual pathway of lung development, provided it occurs during a critical window of opportunity before the end of somatic growth. During this period, the mechanical constraints of the water and intermittent apneas drive an unparalleled adaptive structural response of the lungs. Furthermore, Paramita et al. (2020) emphasized the importance of structured exercise periodization, finding significant positive correlation between formalized training programs, maximized lung function, and competitive achievements in young adult swimmers. Furthermore, the specific breathing pattern of swimming—characterized by prolonged breath-holding and restricted ventilation - serves as a unique hypertrophic trigger. As highlighted by Basavaraj et al. (2014) and supported by Nicoară et al. (2024), this inherent restriction momentarily causes intermittent hypoxia, which forces the body into anaerobic metabolism and potently stimulates the respiratory center in the medulla. The chronic exposure to this intermittent hypoxia, combined with the mechanical stretch of apnea exercises, is theorized to promote alveolar hyperplasia (an actual increase in the number of alveoli), thereby physically expanding the vital capacity of the lungs.

The Role of Supplemental Respiratory Training

While the act of swimming itself serves as a powerful stimulus for lung expansion, researchers have also explored the efficacy of supplemental respiratory and resistance training. Vašičková et al. (2017) found that incorporating one month of dedicated respiratory muscle training (RMT), using positive expiratory pressure and inspiratory muscle trainers, significantly improved inspiratory muscle strength and maximal underwater swimming distance (apnea) in young fin-swimmers. Conversely, for highly trained elite athletes, the aquatic environment itself may provide a maximal conditioning stimulus to the respiratory musculature. Mickleborough et al. (2008) demonstrated that combining a rigorous 12-week competitive swim training program with specific inspiratory muscle training (IMT) resulted in no additional enhancements in pulmonary efficiency or respiratory muscle strength compared to swim training alone. This highlights that intensive swimming inherently imposes such a high resistive load on respiration that supplemental inspiratory muscle training devices may offer negligible additional benefits for already elite swimmers.

Furthermore, not all forms of dry-land resistance training directly translate to improved lung capacity. Yapıcı-Öksüzoğlu (2020) investigated the effects of a 6-week Theraband training program performed in addition to standard swim training. While the experimental group improved in upper extremity strength, there were no statistically significant differences in respiratory parameters (such as FVC and FEV1) between the experimental and control groups. This indicates that standard aquatic exercise might already maximize basic spirometric gains, and dry-land resistance training does not further push respiratory limits.

Taken together, these studies indicate that swimming exerts a uniquely potent hypertrophic stimulus on the respiratory system, significantly expanding static and dynamic lung volumes through the mechanical resistance of water and controlled breathing patterns.

Comparison with Other Athletes

The respiratory adaptations observed in competitive swimmers frequently exceed those seen in both sedentary individuals and athletes participating in land-based sports. A substantial body of literature has been dedicated to comparing the pulmonary function of swimmers with other elite athletes, exploring variables such as static and dynamic lung volumes, gas diffusion capacities, breathing mechanics, and respiratory health.

Static and Dynamic Lung Volumes

A consistent finding across multiple comparative studies is that swimmers possess significantly larger static lung volumes, particularly Forced Vital Capacity (FVC) and Vital Capacity (VC), when compared to land-based athletes. Lazovic-Popovic et al. (2016) conducted a large-scale study comparing 38 elite male swimmers with 271 elite football players and 100 sedentary controls. The results demonstrated that swimmers had statistically higher values for VC, FVC, and Forced Expiratory Volume in 1 second (FEV1) compared to both the football players and the sedentary group. Interestingly, the football players and sedentary controls showed no significant differences between each other in these parameters, suggesting that the standard aerobic demands of land-based sports like football may not be sufficient to induce the pulmonary hypertrophy seen in swimmers. However, their data also revealed that swimmers possessed a significantly lower FEV1/FVC ratio (80.6%) compared to football players (85.9%) and controls (84.5%), reflecting a physiological phenomenon where the substantial expansion of vital capacity outweighs the proportional increase in airway caliber.

The disparity is further supported by research examining athletes across a wider variety of sports. Bernhardsen et al. (2023) investigated Norwegian national-team athletes from different disciplines, including swimmers, cross-country skiers, speed-skaters, rowers/paddlers, handball and soccer players. Their findings indicated that the mean FVC Z-score was the highest among swimmers compared to all other sports. Additionally, mean FEV1 values were higher than the Global Lung Initiative (GLI) reference values for both swimmers and ball game athletes. In contrast, endurance athletes, including swimmers, skiers, and rowers, exhibited lower than reference values for forced expiratory flow (FEF25-75) and the FEV1/FVC ratio, aligning with the concept of disproportional lung volume expansion. To comprehensively compare the respiratory system across disciplines, one must evaluate not only lung volumes but also the dynamic strength of the respiratory pump. A recent extensive profiling of Olympic-level athletes by Kowalski et al. (2025) utilizing the S-Index test revealed that elite swimmers exhibit exceptionally high dynamic inspiratory muscle strength. In their cohort, male swimmers generated S-Index values (ranging up to 181.7 cmH2O) that were among the highest of all evaluated sports, paralleling or slightly exceeding other elite water-based endurance athletes like rowers, and significantly surpassing land-based athletes, e-sports competitors, and age-matched controls. This indicates that the aquatic environment forces a near-maximal functional adaptation of the inspiratory muscle force, distinguishing swimmers from terrestrial athletes.

Rosser-Stanford et al. (2019) similarly observed that daily active swimmers had significantly larger FVC (109% of predicted values) than matched field sport athletes (rugby and football, 106% of predicted values) and recreationally active adults (99% of predicted values). However, unlike the findings of Lazovic-Popovic et al. (2016), Rosser-Stanford et al. (2019) did not find any significant differences in FEV1 or Maximal Voluntary Ventilation (MVV) between the groups.

Because the lung volume is strongly correlated with anthropometric features like height and body mass, some researchers have utilized allometric scaling to ensure that the observed superiority of swimmers' lungs is not merely a byproduct of them being taller or larger. Doherty and Dimitriou (1997) evaluated 459 asymptomatic Greek children and young adults, categorizing them into swimmers, land-based athletes, and sedentary controls. Using logarithmic transformations and allometric equations to control for stature and age, they confirmed that both male and female swimmers possessed a larger FEV1 independent of body size compared to both land-based athletes and sedentary controls.

Interestingly, while the majority of literature supports the superiority of swimmers’ lung volumes, a recent study by Rodrigues et al. (2025) comparing male swimmers, triathletes, and runners did not find significant cross-sport differences in lung function variables under various testing conditions (seated on land, prone on land, seated immersed and prone immersed). Nevertheless, they found that the accumulated years of swim training were significantly correlated with higher FVC and FEV1 measurements specifically during the immersed seated condition, reinforcing that long-term exposure to the aquatic environment drives specific pulmonary adaptations.

Comparisons Within Aquatic Disciplines

Differences in lung function exist not only between aquatic and terrestrial athletes but also among different types of water-based sports. García et al. (2021) compared elite aquatic athletes, dividing them into swimmers, artistic swimmers, and water polo players. They found that male swimmers exhibited significantly higher values for FVC (6.11 L vs. 4.87 L) and FEV1 (4.96 L vs. 4.10 L) than male water polo players. Furthermore, male swimmers presented higher maximum oxygen consumption ($\dot{V}$O2max) and maximum ventilation ($\dot{V}$Emax) than their water polo counterparts. Among the female cohorts, swimmers showed higher $\dot{V}$O2max values than both artistic swimmers and water polo players, although there were no significant differences between the female groups regarding FVC and FEV1.

Furthermore, physiological superiority is evident not only when comparing different water-based sports, but also when comparing different performance tiers within swimming itself. In their allometric scaling study, Doherty and Dimitriou (1997) highlighted that national standard (elite) swimmers possessed significantly larger lung volumes, especially FEV1, compared to non-national standard (sub-elite) swimmers. This appears to be crucial instance demonstrating that maximal pulmonary development is a distinguishing characteristic of the most successful competitors, even among trained swimmers.

Breathing Mechanics and Gas Exchange

The enhanced static volumes of swimmers translate into distinct ventilatory strategies and gas exchange efficiencies during exercise. Rosser-Stanford et al. (2019) found that during maximal exertion on a cycle ergometer, swimmers, field sport athletes, and recreational controls all utilized roughly the same portion of their MVV and FVC maintaining a similar ventilatory reserve. However, the swimmers achieved a significantly greater peak minute ventilation (VE peak) than the recreational group, and crucially, they delivered this volume by adopting an alternative breathing strategy: breathing slower and more deeply (higher tidal volume, VT) than the non-swimmers.

Furthermore, the aquatic environment and swimming mechanics have a profound effect on pulmonary alveoli-capillary diffusion. García et al. (2021) measured the diffusion capacity of carbon monoxide (DLCO) in aquatic athletes and discovered that DLCO was exceptionally high, far exceeding the reference values for their age and height (135% of predicted for females and 148% of predicted for males). While male swimmers had a higher DLCO than water polo players, no differences in diffusion capacity were observed between female swimmers, artistic swimmers, and water polo players. The superior diffusion capacity (DLCO) seen in swimmers appears to be structurally driven by the sheer size of the expanded alveolar space. García et al. (2021) demonstrated that male swimmers not only possess higher DLCO but also a significantly larger Alveolar Volume (VA) compared to male water polo players (8.36 L vs. 6.94 L). When the diffusion capacity was mathematically corrected for alveolar volume (KCO), the differences between the aquatic disciplines were minimized. This confirms that the unparalleled gas exchange efficiency in swimmers is predominantly a function of their massively expanded alveolar surface area - achieved through specific swimming kinematics - rather than a change in the intrinsic permeability of the alveolar-capillary membrane.

This superior gas transfer may be attributed to a larger alveolar surface area and the redistribution of blood flow caused by horizontal positioning and hydrostatic pressure.

Respiratory Health and Pathophysiological Paradox

Despite possessing superior lung capacities and diffusion rates, competitive swimmers often suffer from a paradoxically high prevalence of respiratory symptoms when compared to other athletes. Bernhardsen et al. (2023) note that bronchial hyperresponsiveness (BHR), a condition linked to asthma and airway inflammation, was highly prevalent among endurance athletes, but it was particularly prominent in swimmers. Depending on the methacholine provocation cut-off used, BHR was observed in 50-87% of the elite swimmers, compared to 25-47% in cross-country skiers, and 0-17% in ball game athletes. Additionally, 83% of the swimmers reported exercise-induced respiratory symptoms such as cough and chest tightness. However, a deeper analysis of the clinical profile presents a fascinating paradox regarding the etiology of this hyperresponsiveness. While Bernhardsen et al. (2023) found the highest rate of severe BHR in swimmers, their data simultaneously revealed that swimmers had the lowest prevalence of systemic allergic sensitization. Specifically, only 26% of elite swimmers had a positive skin prick test (atopy), compared to 37.5% of cross-country skiers and over 50% of rowers and speed skaters. This stark immunological contrast confirms that the airway hyperresponsiveness observed in swimmers is primarily a non-allergic, occupationally induced mechanical and chemical injury driven by the pool environment, rather than a classic atopic disease profile common in the general population.

Similarly, the exposure to swimming-specific environments (like chlorinated pools) forces adaptations in allergic or asthmatic athletes. Rodrigues et al. (2025) noted that while swim training primarily enhances FVC in healthy athletes, for allergic and asthmatic athletes, the training predominantly promotes improvements in dynamic variables like the FEV1/FVC ratio and FEF25-75%. This highlights that while swimmers outperform terrestrial athletes in structural lung volumes, their airways are concurrently subjected to significant chemical and mechanical stress that often manifests as hyperresponsiveness and sports-induced asthma.

This paradox is not limited to regional cohorts but is strikingly evident on a global scale. Mountjoy et al. (2015) analyzed Therapeutic Use Exemptions (TUEs) for asthma medication across multiple Olympic Games and FINA World Championships. Their extensive data conclusively demonstrated that athletes in endurance aquatic disciplines - specifically swimming, open water swimming, synchronized swimming, and water polo - exhibited a statistically higher prevalence of asthma and airway hyperresponsiveness compared to athletes in non-endurance aquatic disciplines (such as diving) and non-endurance Olympic sports overall.

Mechanisms of Adaptation

The remarkable pulmonary capacities observed in competitive swimmers are not merely a byproduct of general aerobic conditioning but are the result of specific, multifaceted physiological and biomechanical adaptations to the aquatic environment. The mechanisms driving these adaptations can be broadly categorized into mechanical constraints of water immersion, hemodynamic fluid shifts, altered ventilatory patterns, and potential structural growth of the lung tissue itself. Understanding these underlying mechanisms is crucial for explaining the disparities in lung function between swimmers and land-based athletes.

Hydrostatic Pressure and the Work of Breathing

The most immediate and continuous stimulus applied to the respiratory system during swimming is water immersion. As highlighted by Leahy et al. (2019), the thorax undergoes unique conditions while swimming, primarily due to the hydrostatic pressure exerted by the surrounding water. Because water is significantly denser than air, it places a substantial external compressive load on the chest wall and abdomen. Consequently, inspiration during swimming is no longer a predominantly passive or low-resistance action; it becomes strenuous, active process where the inspiratory muscles (primarily the diaphragm and external intercostals) must generate sufficient force to overcome both the elastic recoil of the lungs and the external water pressure.

The continuous resistive loading acts as a form of highly specific, sport-induced respiratory muscle training (RMT). Over years of intense practice, the inspiratory muscles undergo significant functional enhancement and resistance to fatigue. Furthermore, Leahy et al. (2019) emphasize that the overall work of breathing (Wb) is considerably higher during swimming compared to equivalent exercise on land, forcing a profound functional adaptation of the respiratory pump. While this muscular conditioning helps swimmers efficiently manage the increased Wb, current literature suggests that the elevated Forced Vital Capacity (FVC) observed in aquatic athletes is driven more by structural adaptations and disproportionate chest wall growth rather than solely by increased static inspiratory muscle strength (Rochat et al., 2022). The magnitude of this mechanical loading cannot be overstated. Research indicates that even at submaximal intensities, such as swimming at 90-95% of race pace, the respiratory muscles are pushed to the point of inspiratory muscle fatigue (IMF) (Mickleborough et al., 2008). This indicates that the aquatic environment imposes a continuous, near-maximal conditioning stimulus on the diaphragm and external intercostals, comparable to high-intensity resistance training. Consequently, the chronic hypertrophy and enhanced fatigue resistance of these muscles are necessary functional adaptations to maintain alveolar ventilation when both metabolic demand and external resistive loads are at their peak.

Hemodynamic Shifts and Lung Compliance

In addition to external compression, the horizontal body posture adopted during swimming, combined with the hydrostatic pressure gradient, induces significant internal hemodynamic changes. Immersion leads to a cephalad (upward) redistribution of venous blood from the lower extremities into the thoracic cavity. This central venous engorgement directly impacts pulmonary mechanics by decreasing lung compliance, meaning the lungs become stiffer and require even greater transpulmonary pressure to inflate (Leahy et al., 2019; Hoshi et al., 2025).

Päivinen et al. (2021) further observed that the transition from seated position on land to a prone swimming position in water acutely alters pulmonary function, significantly affecting forced expiratory volume and flow rates. The respiratory system must adapt to maintain adequate minute ventilation (VE) despite these restrictive and resistive constraints. Furthermore, these hemodynamic shifts do not merely restrict lung compliance; they simultaneously offer a profound physiological advantage regarding gas exchange. The cephalad redistribution of venous blood significantly engorges the pulmonary capillary bed (Pendergast et al., 2015). As García et al. (2021) point out, this chronic increase in pulmonary capillary blood volume, combined with mechanical stretching, maximizes the alveolar surface area available for gas diffusion. This physiological mechanism explains the exceptionally high lung diffusion capacity for carbon monoxide (DLCO) observed in elite swimmers, which frequently exceeds 130-150% of predicted reference values. Therefore, the aquatic environment physically forces an optimization of the alveoli-capillary membrane, enhancing the efficiency of oxygen uptake during periods of intense aerobic demand.

Ventilatory Patterns and Biomechanics

The mechanics of the swimming stroke inherently dictate respiratory frequency and timing. Unlike runners or cyclists who can freely adjust their breathing rate to match their metabolic demands, swimmers face a mechanically restricted breathing pattern. Inspiration can only occur during specific, brief phases of the stroke cycle when the face clears the water. This forces swimmers to adopt a strategy characterized by controlled-frequency breathing, necessitating the intake of very large tidal volumes (VT) in exceptionally short inspiratory times (Leahy et al., 2019; Päivinen et al., 2021).

The repeated pattern of breath-holding (apnea) interspersed with explosive exhalations and rapid, maximal inhalations places extreme mechanical stretch on the lung parenchyma. Furthermore, this breathing strategy often induces transient states of mild hypoxia and hypercapnia, which act as potent chemical stimuli for respiratory drive and may trigger long-term compensatory adaptations at the cellular level (Rochat et al., 2022). The retainability of a horizontal posture is also critical. Watanabe et al. (2017) and Moriyama et al. (2021) note that minimizing the distance between the center of buoyancy (CoB) and center of mass (CoM) reduces sinking torque and drag. A higher lung volume physically shifts the CoB cranially, providing a biomechanical advantage by elevating the legs and improving swimming economy. Therefore, expanding lung volume is not only a physiological adaptation but a biomechanical necessity for elite performance.

The Structural vs. Functional Debate (Alveolar Hyperplasia)

A central question in sports pulmonology is whether the increased lung volumes in swimmers are purely functional (due to stronger respiratory muscles and improved chest wall compliance) or structural (due to actual physical growth of the lungs). Rochat et al. (2022) comprehensively reviewed the determinants of lung function changes in athletic swimmers, pointing out that while intense training undeniably strengthens respiratory muscles, there is compelling evidence suggesting structural adaptations, particularly if training begins early in life.

The intense mechanical stretching of the lungs during maximal inspirations against hydrostatic pressure, combined with increased pulmonary blood flow, may stimulate alveolar hyperplasia (an increase in the number of alveoli) and enhanced alveolar growth if the stimulus occurs during the critical windows of childhood and adolescent development (Rochat et al., 2022). While the exact molecular pathways remain a subject of investigation, the prevailing theory suggests that the unique combination of hypoxia, mechanical stretch, and high oxygen demand experienced during youth swim training physically alters the architectural development of the lungs, leading to permanently enlarged static lung volumes that persist into adulthood.

Operating Lung Volumes and Dynamic Constraints

To fully comprehend the ventilatory mechanics in swimmers, one must also consider the concept of operating lung volumes (OLV) during immersion. As demonstrated in a recent study by Hoshi et al. (2025), water immersion physically displaces the OLV toward the expiratory side. Specifically, the resting expiratory reserve volume (ERV) significantly decreases due to abdominal compression, while the inspiratory reserve volume (IRV) increases compared to land-based environments. During aquatic exercise, swimmers must continuously adjust their tidal volume (VT) within this restricted, shifted operating space. This mechanical displacement forces the respiratory system to adapt to breathing at lower absolute lung volumes, which alters the elastic recoil properties of the chest wall and forces the diaphragm to operate at a different length-tension relationship to effectively overcome the hydrostatic pressure. Such daily mechanical constraints further underscore why the respiratory architecture of competitive swimmers fundamentally diverges from that of terrestrial athletes.

Clinical and Health Implications, Environmental Risks

While the aquatic environment provides an exceptional stimulus for the structural and functional enhancement of the respiratory system, it simultaneously presents a unique set of clinical challenges and environmental risks. The literature describes a phenomenon often referred to as the "swimmer's paradox": despite possessing superior static and dynamic lung volumes, competitive swimmers exhibit one of the highest prevalences of airway hyperresponsiveness (AHR), exercise-induced bronchoconstriction (EIB), and asthma among all elite athletes. Understanding the etiology of these conditions - ranging from the mechanical stress of hyperventilation to the chemical toxicity of indoor pool environments – is essential for optimizing both performance and long-term respiratory health.

The Paradox of Elite Swimming: High Prevalence of Asthma and EIB

The prevalence of asthma and AHR in aquatic sports is remarkably high when compared to terrestrial Olympic disciplines. Mountjoy et al. (2015) analyzed the prevalence of asthma and AHR across various aquatic disciplines by tracking Therapeutic Use Exemptions (TUEs) submitted by elite athletes. Their findings underscore that competitive swimming and other aquatic sports subject the airways to intense physiological stress, resulting in a disproportionately high reliance on inhaled beta-2 agonists for asthma management compared to other Olympic sports.

Even among athletes without prior clinical diagnosis of asthma, the incidence of exercise-induced bronchoconstriction (EIB) is alarmingly high. Lima et al. (2024) conducted a comprehensive systematic review and meta-analysis evaluating the prevalence of EIB specifically in non-asthmatic swimmers. Analyzing data from 33 studies encompassing 700 non-asthmatic swimmers, the researchers documented 250 confirmed cases of EIB (an overall prevalence of 34%). This high prevalence firmly establishes that the rigorous demands of competitive swimming are sufficient to induce transient airway narrowing during or immediately following intense exercise, even in the absence of chronic allergic asthma. Furthermore, Eksi et al. (2021) investigated adolescent elite swimmers (aged 10-17) and found that while the prevalence of lower airway EIB was still relatively low at this young age (8.5%, similar to the general population), there was a significant post-exercise increase in nasal discharge and exercise-induced rhinitis. This nuance indicates that while structural lower airway dysfunction (EIB) may require years of cumulative exposure to high training volumes and chlorine to fully manifest, the irritation of the upper respiratory tract begins very early in an athlete's career.

Complicating this paradox is the methodological challenge of accurately diagnosing EIB in aquatic athletes. As Leahy et al. (2020) emphasize, 'context matters' in respiratory testing; the standard laboratory Eucapnic Voluntary Hyperpnea (EVH) test, while considered the gold standard, fundamentally fails to replicate the specific environmental conditions of the pool, such as high humidity and the presence of chloramines. This discrepancy is starkly reflected in the meta-analysis by Lima et al. (2024), which revealed that EIB prevalence varies drastically depending on the diagnostic method used. While the Methacholine Challenge Test (MCT) and EVH detected EIB in 51% and 43% of non-asthmatic swimmers respectively, standard Exercise Challenge Tests (ECT) yielded a prevalence of only 12%. This highlights a critical clinical implication: relying solely on traditional terrestrial exercise protocols may severely underdiagnose airway dysfunction in aquatic athletes.

Environmental Triggers: Chlorine and Disinfection By-Products (DBPs)

The primary environmental risk factor distinguishing swimming from other sports is chronic exposure to the chemical composition of pool water and the air immediately above the water surface. To maintain hygiene, swimming pools are heavily treated with chlorine-based disinfectants. When chlorine reacts with organic matter introduced by swimmers (such as sweat, saliva, and urea), it forms volatile disinfection by-products (DBPs), most notably nitrogen trichloride (trichloramine).

Bernard et al. (2003) provided groundbreaking evidence regarding the toxicity of these DBPs on the respiratory epithelium of children. In their study of 226 healthy schoolchildren, the researchers measured serum biomarkers of lung epithelial integrity, specifically the 16 kDa Clara cell protein (CC16) and surfactant-associated proteins (SP-A and SP-B). They discovered that exposure to nitrogen trichloride in indoor chlorinated pools causes significant lung hyperpermeability. The disruption of the epithelial barrier facilitates the penetration of airborne allergens into the deeper layers of the respiratory tract, unexpectedly associating frequent indoor pool attendance with an increased risk of asthma and allergic sensitization.

Reducing these environmental triggers requires a deeper understanding of the physical properties of indoor pool facilities. As highlighted by Sepioło et al. (2025), trichloramine is a highly volatile compound that is heavier than air, causing it to accumulate precisely in the thin layer of air just above the water surface—the exact 'breathing zone' where swimmers take massive, rapid inhalations. Furthermore, recent field studies demonstrate that standard ASHRAE-compliant ventilation systems (typically providing 4 to 6 air changes per hour) often fail to prevent the accumulation of these DBPs. Implementing advanced air exchange strategies that significantly increase the outside-air fraction has been shown to reduce breathing-zone trichloramine by up to 55%, underscoring that facility management is as critical to respiratory health as medical intervention (Sepioło et al., 2025).

Cellular Damage, Oxidative Stress, and Airway Remodeling

The continuous inhalation of DBPs during periods of massive ventilatory demand (such as maximal exertion in a pool) induces profound cellular and oxidative changes. Škrgat et al. (2018) meticulously evaluated systemic and airway oxidative stress in 41 healthy, non-asthmatic elite swimmers during a period of high-Intensity training preceding a national championship. The researchers analyzed induced sputum and peripheral blood, finding a staggering ten-fold increase in 8-isoprostanes (a potent biomarker of oxidative stress and lipid peroxidation) in the induced sputum. However, contrary to expectations, they found no significant elevation in airway neutrophils or other inflammatory cytokines. These findings highlight that while the chlorinated pool environment directly interacts with the airway epithelium to trigger severe local oxidative stress, this does not necessarily translate into immediate cellular airway inflammation in young, healthy athletes without clinical asthma.

Despite the absence of acute inflammation in some cohorts, chronic exposure to chemical irritants combined with intense mechanical stretch is known to alter airway architecture over time. Silvestri et al. (2013) evaluated pulmonary function and airway responsiveness in young competitive swimmers (aged 7-19 years). They observed that the athletes' lifetime exposure-quantified as the total hours spent training in indoor pools - was strongly correlated with specific spirometric changes. While Forced Vital Capacity (FVC) increased more than expected with normal growth (confirming the hypertrophic effect of swimming), the forced expiratory flows did not increase proportionally, leading to a reduced FEV1/FVC ratio. Crucially, rather than viewing this as an obstructive abnormality linked to AHR, the authors concluded that this disproportionate (dysanaptic) growth between lung volume and airway caliber represents a physiological variant. Furthermore, this structural adaptation occurred irrespective of the presence of allergic sensitization or airway hyperresponsiveness, indicating that the mechanical and environmental stresses of swimming uniquely reshape the respiratory system. A crucial clinical question arising from this extensive airway remodeling is whether the swimmer's asthma phenotype is a permanent structural disease or a transient occupational hazard. Evidence suggests a significant degree of plasticity and reversibility in the respiratory system of these athletes. As noted in the comprehensive epidemiological review by Mountjoy et al. (2015), several prospective and retrospective observations demonstrate that airway hyperresponsiveness can partially or fully reverse following a prolonged rest period (e.g., two weeks out of the water) or upon retirement from competitive swimming. This reversibility supports the hypothesis that the intense oxidative stress and AHR observed in elite swimmers are primarily driven by active, high-volume exposure to chlorinated environments rather than an irreversible, chronic asthmatic condition.

Upper Airway Disorders: Rhinitis in Swimmers

The environmental risks of swimming are not confined to the lower respiratory tract; the upper airways are equally susceptible. A recent comprehensive review by Sepioło et al. (2025) exploring asthma and upper airway disorders in competitive swimmers emphasized the high prevalence of occupational-style rhinitis in this population. The constant flow of chlorinated water through the nasal passages, combined with the inhalation of chloramines, strips the nasal mucosa of its protective mucus layer. Eksi et al. (2021) investigated this phenomenon in elite adolescent swimmers and confirmed a significant post-exercise increase specifically in nasal discharge (rhinorrhea). Interestingly, their objective acoustic rhinometry assessments revealed no statistically significant changes in nasal resistance or congestion. This suggests that while the chlorinated environment provokes a severe secretory response (hypersecretion), the structural patency of the nasal passages remains largely intact. However, the combination of copious nasal discharge and the inherent mechanical demands of the swimming stroke forces athletes to rely heavily on mouth-breathing. This subsequently bypasses the natural filtration and humidification functions of the nose and delivers cold, dry, and chemically laden air directly into the fragile lower airways.

Clinical Implications: Therapeutic Benefits for Asthmatic Youth vs. Elite Risks

Despite the clear occupational hazards faced by elite competitive swimmers, a critical clinical distinction must be made regarding the therapeutic application of swimming for the general pediatric population. While massive, elite-level exposure to chlorinated environments drives AHR, moderate recreational swimming remains one of the most highly recommended physical interventions for children with existing asthma.

Ptak and Szyc (2024) reviewed the impact of swimming training on the course of bronchial asthma, concluding that appropriately dosed aquatic exercise significantly alleviates asthma symptoms and improves overall respiratory system function in non-elite populations. The warm, humid air close to the water surface generally prevents the airway cooling and desiccation that triggers typical exercise-induced asthma on land. Validating this therapeutic approach, Tizar et al. (2023) conducted an experimental study on 20 asthmatic children (aged 6-18 years) who underwent an 8-week swimming program (60 minutes per session, three days a week). The results demonstrated a statistically significant improvement in vital lung capacity and respiratory function tests post-intervention.

Therefore, the clinical implications of swimming are highly dose-dependent. For children and adolescents with asthma, structured, moderate swimming training enhances lung capacity, respiratory muscle strength, and symptom control. Conversely, for elite athletes, the sheer volume of training - often exceeding 20 hours per week in heavily chlorinated indoor environments - tips the physiological scale from adaptation to toxicity, resulting in lung hyperpermeability, oxidative stress, and a paradoxically high prevalence of swimmer's asthma.

Discussion

Summary of Evidence: The Dual Nature of Aquatic Adaptation

This systematic review aimed to synthesize the existing literature on the respiratory adaptations, pulmonary function parameters, and environmental risks associated with competitive swimming. The aggregated evidence conclusively demonstrates that swimming induces profound, sports-specific adaptations in the respiratory system. Across multiple studies, swimmers consistently exhibit superior static and dynamic lung volumes - specifically Forced Vital Capacity (FVC), Vital Capacity (VC), and Forced Expiratory Volume in one second (FEV1) - when compared to land-based athletes and sedentary controls. Furthermore, these adaptations extend to gas exchange efficiency, with swimmers demonstrating exceptionally high diffusion capacity (DLCO) driven by an expanded alveolar surface area.

The mechanisms underpinning these adaptations appear to be a complex interplay of mechanical, hemodynamic, and environmental factors. The continuous exposure to hydrostatic pressure increases the work of breathing, acting as a potent respiratory muscle training stimulus. Moreover, the unique breathing patterns imposed by the swimming stroke, characterized by intermittent hypoxia and massive tidal volumes, may trigger isotropic lung growth during prepuberty and stimulate alveolar hyperplasia. However, the debate regarding whether these superior capacities are purely the result of intensive training (nurture) or genetic predisposition (nature) remains partially unresolved, although significant correlations between accumulated training years and lung volume expansion strongly support the adaptative hypothesis.

The “Swimmer’s Paradox” and Environmental Toxicity

A central finding of this review is the prominent "swimmer's paradox." While the aquatic environment promotes maximal lung development, it concurrently exposes the airways to severe chemical stress. The literature robustly confirms that elite swimmers present with a disproportionately high prevalence of airway hyperresponsiveness (AHR), exercise-induced bronchoconstriction (EIB), and asthma. Crucially, this review highlights that the etiology of "swimmer's asthma" fundamentally differs from classic atopic asthma.

Chronic inhalation of disinfection by-products (DBPs), most notably nitrogen trichloride (trichloramine) present in indoor pools, disrupts the lung epithelial barrier, leading to increased lung hyperpermeability. This chemical injury, combined with the mechanical stress of extreme hyperventilation, induces massive local oxidative stress, as evidenced by significantly elevated 8-isoprostane levels in the airways of elite swimmers. Interestingly, this oxidative stress does not always translate into classic neutrophilic or eosinophilic inflammation, suggesting a unique, occupationally induced pathophysiological pathway. The clinical implications of this are highly dose-dependent: while moderate swimming remains a highly recommended, therapeutic exercise for asthmatic children, the sheer volume of exposure in elite aquatic athletes tips the scale toward respiratory toxicity.

Methodological Challenges and Diagnostic Limitations

The synthesis of the included studies revealed significant methodological challenges in evaluating airway dysfunction in aquatic athletes. Diagnosing EIB in swimmers is particularly problematic because traditional clinical protocols often fail to replicate the specific environmental triggers of the pool. As demonstrated in recent literature, "context matters"; standard laboratory Eucapnic Voluntary Hyperpnea (EVH) tests or terrestrial exercise challenges may severely underdiagnose EIB because they lack the high humidity and chloramine exposure characteristic of the swimming environment. This discrepancy is reflected in varying EIB prevalence rates depending on the diagnostic method utilized, with the Methacholine Challenge Test (MCT) often resulting in higher positive rates than standard exercise tests. Consequently, relying on standardized land-based diagnostic criteria may be insufficient for this specific athletic population.

Limitations of the Current Literature

While this systematic review provides a comprehensive overview, several limitations within the primary literature must be acknowledged. First, the majority of the included studies are cross-sectional or retrospective in design. This limits the ability to establish definitive causality between swim training and structural lung growth, as the influence of natural selection (i.e., individuals with genetically larger lungs succeeding and remaining in the sport) cannot be entirely ruled out. Second, studies often fail to adequately quantify the exact concentration of DBPs during training sessions, relying instead on surrogate markers like pool hall height or total hours spent in the water. Finally, variations in anthropometric scaling remain an issue; while higher-quality studies utilize allometric scaling to control for stature and age, many studies still rely on simple ratios, which may misrepresent true physiological differences.

Future Research Directions and Practical Implications

Future research must prioritize long-term, longitudinal studies tracking respiratory function in swimmers from prepuberty through adulthood to definitively isolate the effects of training from genetic endowment. Additionally, there is a critical need to develop and validate sport-specific diagnostic challenge tests that incorporate DBP exposure to accurately assess EIB in aquatic athletes.

From a practical standpoint, the findings of this review underscore the necessity for a paradigm shift in swimming pool management and athlete care. Facility managers must implement advanced air exchange and ventilation systems designed specifically to extract heavy, volatile chloramines from the "breathing zone" just above the water surface. For clinicians and coaches, routine screening for epithelial damage and oxidative stress, rather than relying only on symptomatic reporting, should become standard practice in elite aquatic sports to protect the long-term respiratory health of the athletes.

Conclusions

This systematic review consolidates the evidence that competitive swimming uniquely reshapes the human respiratory system, creating a profound physiological contrast. On one hand, the aquatic environment and the biomechanical demands of the swimming stroke act as unparalleled stimuli for pulmonary development. Chronic exposure to hydrostatic pressure, intermittent hypoxia, and the mechanical necessity of inhaling massive tidal volumes drive structural adaptations such as isotropic lung growth and alveolar hyperplasia. Consequently, elite swimmers are endowed with supranormal static and dynamic lung capacities, as well as an exceptionally efficient alveolar-capillary diffusion, which clearly exceed the physiological limits observed in terrestrial athletes.

On the other hand, these remarkable morphological advantages are counterbalanced by the "swimmer's paradox". The rigorous ventilatory demands of competitive swimming, combined with the chronic inhalation of volatile disinfection by-products (DBPs) such as trichloramine, inflict significant mechanical and chemical stress on the airway epithelium. This occupational hazard manifests as a remarkably high prevalence of exercise-induced bronchoconstriction (EIB), airway hyperresponsiveness, and a non-allergic asthma phenotype among elite aquatic athletes.

Crucially, the impact of swimming on respiratory health is highly dose-dependent. While moderate, recreational swimming remains a highly effective and recommended therapeutic intervention for improving lung function and symptom control in asthmatic youth, the extreme training volumes of elite competitors tip the physiological balance toward respiratory toxicity.

To safeguard the long-term health of these athletes, a multidisciplinary approach is essential. Clinicians must move beyond standard terrestrial diagnostic protocols, utilizing sport-specific challenges (such as the Methacholine Challenge Test or pool-environment testing) to avoid underdiagnosing airway dysfunction in swimmers. At the same time, facility managers and engineers must prioritize advanced ventilation and water purification strategies to minimize chloramine accumulation in the breathing zone just above the water surface. Ultimately, maximizing the profound physiological benefits of swimming while alleviating its environmental risks remains the foremost challenge in modern aquatic sports medicine.

Ethical Considerations Plagiarism: The manuscript represents original work and has not been published elsewhere.

Ethics Approval: As this study is a systematic literature review synthesizing previously published research, ethical approval from an institutional review board was not required.

Conflicts of Interest: The authors declare no conflicts of interest.

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