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# IMPACT OF AIR POLLUTION ON ATHLETIC PERFORMANCE AND HEALTH: A COMPREHENSIVE REVIEW

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**ABSTRACT**

Air pollution represents a major global health challenge and has been increasingly recognized as an important environmental factor influencing respiratory and cardiovascular function as well as athletic performance. During exercise, elevated minute ventilation and oral breathing increase inhaled pollutant dose and modify particle deposition patterns within the respiratory tract. Particulate matter ($PM_{2.5}$ and $PM_{10}$), nitrogen oxides, ozone, sulfur dioxide, and carbon monoxide are among the most relevant pollutants affecting exercise physiology.

Inhaled pollutants may induce oxidative stress, systemic inflammation, endothelial dysfunction, and autonomic imbalance. Biological effects are mediated through reactive oxygen species generation, inflammatory cytokine activation, and potential translocation of ultrafine particles across the alveolar-capillary barrier. In athletes, these mechanisms may lead to transient reductions in pulmonary function, bronchial hyperresponsiveness, decreased maximal oxygen uptake ($VO_2max$), and impaired exercise performance.

Although short-term exposure may cause acute functional impairment, regular physical activity remains beneficial for health. Monitoring air quality, adjusting training intensity, and optimizing exercise timing are recommended preventive strategies. Further prospective studies are needed to evaluate long-term exposure effects on athletic adaptation and cardiovascular outcomes.

**KEYWORDS**

Air Pollution, Athletic Performance, Health Effects, Particulate Matter, Endurance Exercise

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## 1. Introduction

Air pollution constitutes one of the most significant global health threats of the 21st century. According to the World Health Organization (WHO), exposure to polluted air is responsible for approximately 7 million premature deaths annually worldwide, primarily due to respiratory and cardiovascular diseases [1]. Fine particulate matter with an aerodynamic diameter below 2.5 μm ($PM_{2.5}$), nitrogen oxides, tropospheric ozone, and other gaseous pollutants are recognized as major risk factors for the development of chronic diseases. Importantly, adverse health effects have been observed even at concentrations below current environmental regulatory standards [2,10].

Physical activity, particularly of an endurance nature, is associated with a substantial increase in minute ventilation and respiratory rate, leading to greater inhalation of airborne pollutants. During high-intensity exercise, oral breathing bypasses the physiological filtration mechanisms of the nasal cavity, facilitating deeper penetration of particulate matter and gaseous pollutants into the lower respiratory tract and potentially into systemic circulation. Previous studies have demonstrated that exposure to air pollutants may lead to impaired pulmonary function, induction of oxidative stress, systemic inflammatory responses, and adverse alterations in cardiovascular function. These mechanisms may translate into reduced physical performance, decreased maximal oxygen uptake ($VO_2max$), shortened time to exhaustion, and overall deterioration in athletic performance [4,23].

### 1.1 Methods

This study was conducted as a narrative review of the literature. Original research articles, review articles, and meta-analyses were considered for inclusion. Studies were included if they examined the effects of air pollution exposure on health outcomes or physical performance in physically active individuals or athletes, with particular attention to respiratory, cardiovascular, and aerobic capacity parameters.

## 2. Characteristics of Air Pollutants

The most important air pollutants of health relevance include particulate matter ($PM_{10}$ and $PM_{2.5}$), nitrogen oxides ($NO_2$), sulfur dioxide ($SO_2$), tropospheric ozone ($O_3$), and volatile organic compounds (VOCs)[10].

Particulate matter $PM_{10}$ and $PM_{2.5}$ differ in aerodynamic diameter, which determines their capacity to penetrate the respiratory tract. Fine particles ($PM_{2.5}$), due to their small size, can reach the alveolar regions of the lungs and subsequently translocate into the systemic circulation. During physical exertion, increased minute ventilation and the predominance of oral breathing facilitate deeper deposition of these particles within the lower respiratory tract[5,10].

Tropospheric ozone, formed through photochemical reactions involving sunlight and precursor pollutants, is a strong oxidizing agent that may cause irritation of the respiratory mucosa and impairment of pulmonary function. Nitrogen oxides and sulfur dioxide also exhibit irritative and pro-inflammatory properties, particularly under conditions of prolonged or repeated exposure[6].

## 3. Biological Mechanisms Underlying the Effects of Air Pollution

Inhaled particulate matter and gaseous pollutants may initiate the generation of reactive oxygen species (ROS), leading to structural and functional damage to airway epithelial cells. Air pollution–induced oxidative stress promotes the activation of inflammatory mediators, including cytokines and chemokines, which may contribute to the development of local and systemic inflammatory responses[7]. In athletes undergoing intensive training, these processes may overlap with exercise-induced physiological inflammation, potentially impairing recovery and adaptive processes[8].

Air pollutants also affect cardiovascular function through endothelial dysfunction, reduced nitric oxide bioavailability, increased blood viscosity, enhanced platelet activation, and alterations in autonomic nervous system regulation, including reduced heart rate variability[3]. These mechanisms may impair exercise tolerance, decrease oxygen delivery efficiency, and increase the risk of adverse cardiovascular events, particularly during high-intensity exertion[3,9].

Furthermore, exposure to polluted air may result in impaired pulmonary function, reduced lung volumes, and increased bronchial hyperresponsiveness[7]. In endurance athletes, for whom respiratory efficiency is a critical determinant of performance, such alterations may significantly influence competitive outcomes.

### 3.1 Biomarkers of Exposure and Biological Effect in Athletes

Air pollution exerts its biological effects through complex mechanisms involving oxidative stress, systemic inflammation, endothelial dysfunction, and alterations in autonomic regulation. The assessment of biomarkers enables the detection of subclinical biological responses to pollutant exposure and provides mechanistic insight into the pathways linking air pollution with impaired respiratory and cardiovascular function [28,30].

#### 3.1.1 Oxidative Stress Biomarkers

Oxidative stress represents a central mechanism underlying the health effects of particulate matter ($PM_{2.5}$) and gaseous pollutants. Fine particles can generate reactive oxygen species (ROS) directly through their surface properties or indirectly via inflammatory cell activation. Increased oxidative burden leads to lipid peroxidation, protein modification, and DNA damage [2,29].

Commonly investigated biomarkers of oxidative stress include: Malondialdehyde (MDA) – a by-product of lipid peroxidation reflecting oxidative damage to cell membranes [28]; 8-hydroxy-2'-deoxyguanosine (8-OHdG) – a marker of oxidative DNA damage [29].; Isoprostanes – indicators of free radical–mediated lipid peroxidation [30,31].

Experimental and panel studies have demonstrated associations between short-term exposure to particulate matter and increased systemic oxidative stress markers, supporting a causal role of ROS in pollution-related pathophysiology [30].

#### 3.1.2 Inflammatory Biomarkers

Air pollution exposure activates innate immune pathways and promotes the release of pro-inflammatory cytokines. Systemic inflammation is considered a key mediator linking pollutant exposure to cardiovascular and respiratory outcomes.

Frequently assessed inflammatory biomarkers include: C-reactive protein (CRP) – a marker of low-grade systemic inflammation [33]; Interleukin-6 (IL-6) – a pro-inflammatory cytokine associated with cardiovascular risk [27]; Tumor necrosis factor-alpha (TNF-α) – involved in endothelial activation and inflammatory signaling; Fractional exhaled nitric oxide (FeNO) – a non-invasive marker of airway inflammation [32].

Population-based analyses have reported positive associations between ambient $PM_{2.5}$ concentrations and elevated CRP and IL-6 levels, suggesting systemic inflammatory activation even at relatively low exposure levels [16,31]. Studies examining airway responses have shown increased FeNO following exposure to traffic-related air pollutants, indicating localized respiratory inflammation [32].

#### 3.1.3 Endothelial and Hemostatic Biomarkers

Air pollution has also been linked to vascular dysfunction and prothrombotic changes. Endothelial injury represents an early step in the development of cardiovascular disease [22].

Relevant biomarkers include: 1) Von Willebrand factor (vWF) – reflecting endothelial activation [29]. 2) Soluble CD62P and soluble CD40 ligand (sCD40L) – markers of platelet activation. Markers of coagulation activation [29].

Controlled exposure studies have demonstrated changes in vascular and coagulation-related biomarkers following exposure to traffic-derived particles, supporting the hypothesis that air pollution contributes to endothelial dysfunction and cardiovascular risk [22, 33].

**3.1.4 Relevance of Biomarkers in Athletic Populations**

In athletes, the interpretation of pollution-related biomarkers requires consideration of exercise-induced physiological responses. Physical exertion itself transiently increases oxidative stress and inflammatory markers. However, the markedly elevated minute ventilation during moderate- and high-intensity exercise leads to increased inhaled pollutant dose, potentially amplifying biological responses compared with sedentary individuals [34].

Recent expert consensus in sports medicine emphasizes that environmental exposure should be integrated into athlete health monitoring, particularly in endurance disciplines characterized by prolonged outdoor training [27].

Thus, biomarker assessment may provide valuable insight into early, subclinical effects of pollution in athletes and help differentiate between adaptive exercise-related changes and environmentally induced inflammatory or oxidative responses [29].

## 4. Deposition of Airborne Particles in the Respiratory Tract During Exercise

Deposition of airborne particulate matter (PM) in the respiratory tract is determined by particle aerodynamic diameter, hygroscopic properties, airway geometry, and ventilatory pattern. The International Commission on Radiological Protection (ICRP) deposition model and subsequent refinements indicate that coarse particles ($PM_{10}$) deposit predominantly in the extrathoracic region, fine particles ($PM_{2.5}$) in the tracheobronchial and alveolar regions, while ultrafine particles (<0.1 μm) demonstrate high alveolar deposition efficiency due to diffusion-driven transport [5,21].

Three principal physical mechanisms govern deposition: Inertial impaction – dominant for particles >5 μm in upper airways; Gravitational sedimentation – significant in small bronchi and bronchioles for particles 1–5 μm; Brownian diffusion – primary mechanism for ultrafine particles in the alveolar region [21].

During exercise, minute ventilation (VE) increases substantially as a result of elevated tidal volume and respiratory frequency. This alters both total inhaled dose and regional deposition patterns. Increased tidal volume enhances peripheral penetration of particles, shifting deposition from central to distal airways [12,14]. Oral breathing during moderate-to-high intensity exercise reduces nasal filtration efficiency, increasing the fraction of particles reaching the lower respiratory tract [7,14]

Experimental and observational data indicate that exercise not only increases inhaled pollutant dose but also modifies deposition fraction. Higher inspiratory flow rates enhance inertial impaction in proximal airways, while prolonged inspiratory time and deeper ventilation facilitate sedimentation and diffusion in distal regions [12,14].

Fine and ultrafine particles deposited in the alveoli interact directly with the alveolar-capillary interface. Ultrafine particles may translocate across the epithelial barrier into systemic circulation, either via paracellular transport or macrophage-mediated mechanisms [8,19,20]. This provides a mechanistic link between pulmonary deposition and extrapulmonary cardiovascular effects.

In endurance athletes, repeated high-ventilation exposure may amplify cumulative alveolar deposition. Studies demonstrate activation of epithelial and inflammatory pathways in athletes training under polluted conditions, suggesting that deposition-related epithelial stress is biologically relevant even in young, otherwise healthy individuals [16,32,34].

Importantly, deposition is not synonymous with clearance. While mucociliary transport removes particles from central airways, alveolar clearance relies primarily on macrophage phagocytosis, a slower process. Persistent alveolar deposition may therefore sustain low-grade inflammation and oxidative stress [2,5].

Collectively, exercise modifies both the magnitude and distribution of particle deposition within the respiratory tract, increasing distal particle penetration due to elevated minute ventilation, oral breathing, and altered airflow dynamics.[8,14,20]. Although exercise increases particle deposition, biological impact also depends on mucociliary clearance efficiency, exposure duration, and individual susceptibility factors.

## 5. Effects of Air Pollution on Physical Performance and Athletic Outcomes

Experimental and observational studies suggest that exposure to air pollution may adversely affect aerobic performance parameters, including maximal oxygen uptake ($VO_2max$), time to exhaustion, and running economy[10,25]. One proposed mechanism involves impaired oxygen transport related to carbon monoxide exposure, which binds to hemoglobin to form carboxyhemoglobin and reduces its oxygen-carrying capacity. Consequently, maximal oxygen uptake may decrease, and endurance exercise duration may be shortened[8].

Combustion-related pollutants and traffic-derived emissions may induce oxidative stress and inflammatory responses within the respiratory and vascular systems, potentially leading to reduced exercise capacity[8].

An increasing number of studies utilize data from real-world competitive events to evaluate the impact of air quality on athletic outcomes. Analyses of elite distance runners have demonstrated that higher exposure to $PM_{2.5}$ and ozone during the preparatory training period was associated with modest but statistically significant impairments in 5-km race performance (approximately 1–1.5%)[11], a magnitude that may be decisive in elite-level competition[11,14].

## 6. Effects of Air Pollution on the Respiratory System in Athletes

The respiratory system plays a central role in exercise adaptation, particularly in endurance sports where efficient oxygen transport determines overall physical performance capacity. Athletes training outdoors are exposed to increased levels of air pollutants due to both prolonged exercise duration and substantial elevations in minute ventilation. Consequently, the respiratory tract represents one of the primary target organs of environmental pollutants[8,14].

### 6.1 Exercise Ventilation and Pollutant Exposure

During intense physical exertion, minute ventilation may increase up to tenfold compared with resting values, significantly elevating the total inhaled dose of air pollutants[8,12]. In endurance athletes, a substantial proportion of breathing occurs through the mouth, bypassing the physiological filtration and humidification mechanisms of the nasal cavity and facilitating deeper penetration of particulate and gaseous pollutants into the lower respiratory tract [13].

Increased tidal volume promotes the deposition of fine particulate matter ($PM_{2.5}$) in the distal regions of the lungs, where these particles may trigger oxidative stress and inflammatory responses [14]. Even short-term exposure during training sessions may have physiological relevance, particularly in athletes who exercise regularly in environments characterized by elevated pollution levels [14,15].

### 6.2 Impairment of Pulmonary Function and Inflammatory Response

Experimental studies have demonstrated that exposure to ozone and particulate matter during physical exertion leads to transient reductions in spirometric parameters, including forced expiratory volume in one second ($FEV_1$) and forced vital capacity (FVC) [13]. This effect appears to be particularly pronounced in athletes training under conditions of elevated ozone concentrations, where decreases in expiratory capacity and increased perception of dyspnea have been observed[15].

Air pollutants induce a local inflammatory response within the airway epithelium, resulting in increased concentrations of pro-inflammatory cytokines and enhanced epithelial barrier permeability [18]. In highly trained athletes, this response may overlap with the physiological inflammatory processes associated with intense exercise, potentially contributing to persistent airway dysfunction [17,18].

In addition, exposure to air pollution has been associated with increased susceptibility to respiratory infections and greater severity of infectious episodes. Epidemiological data indicate that outdoor air pollution, particularly in urban environments, is linked to a higher burden of acute respiratory infections. Exposure to nitrogen dioxide ($NO_2$) and particulate matter has been associated with increased incidence of laryngotracheobronchitis and viral respiratory infections in European urban populations. Individuals with pre-existing airway inflammation or structural airway alterations may be particularly vulnerable to these effects. Although much of the evidence concerns pediatric populations, the underlying mechanisms—oxidative stress, impaired mucociliary clearance, and dysregulated immune responses—are biologically relevant to athletes exposed to repeated high ventilatory loads in polluted environments[16].

### 6.3 Bronchial Hyperresponsiveness and Respiratory Symptoms

One of the clinically relevant problems observed in athletes training in polluted environments is an increased prevalence of bronchial hyperresponsiveness and symptoms consistent with exercise-induced bronchoconstriction [15]. Epidemiological studies suggest that long-term exposure to traffic-related air pollution may exacerbate exercise-induced bronchospasm, even in individuals without previously diagnosed obstructive airway disease [14].

Athletes participating in winter and endurance sports appear particularly susceptible to these disturbances, likely due to the combined effects of high ventilatory rates, cold and dry air exposure, and environmental pollutants[15]. Symptoms such as cough, wheezing, chest tightness, and dyspnea may impair exercise performance and negatively influence recovery processes [19].

## 7. Effects of Air Pollution on the Cardiovascular System in Physically Active Individuals

In recent years, a growing body of evidence indicates that both short-term and chronic exposure to particulate matter and gaseous pollutants may lead to hemodynamic disturbances, endothelial dysfunction, and an elevated risk of cardiovascular events.[20] In the context of sport, this issue is of particular importance, as intense physical exertion increases the inhaled dose of pollutants and may amplify their systemic effects.

### 7.1 Pathophysiological Mechanisms of Air Pollution Impact on the Cardiovascular System

One of the key mechanisms underlying the cardiovascular effects of air pollution is the induction of oxidative stress and systemic inflammatory responses. Inhalation of $PM_{2.5}$ and gases such as nitrogen dioxide ($NO_2$) and ozone ($O_3$) leads to increased production of reactive oxygen species and the release of pro-inflammatory cytokines[13,21]. These processes contribute to vascular endothelial injury and reduced nitric oxide bioavailability. Human studies suggest that these effects are mediated through pro-inflammatory cytokines such as IL-6, TNF-α, and C-reactive protein (CRP), with elevated levels of IL-6 and CRP being linked to an increased risk of acute myocardial infarction[21,22].

As a consequence, increased vascular tone, impaired blood pressure regulation, endothelial dysfunction, elevated myocardial oxygen demand, and acceleration of atherosclerotic processes may occur[21,22].

Review studies also indicate activation of the sympathetic nervous system during exposure to air pollutants, manifested by increases in blood pressure and alterations in heart rate variability (HRV) [20]. Reduced HRV is recognized as a marker of elevated cardiovascular risk and has been observed even at relatively low concentrations of particulate matter[13,22,].

### 7.2 Acute Cardiovascular Responses During Exercise in Polluted Environments

Carbon monoxide plays a particularly important role in polluted environments, as its binding to hemoglobin limits oxygen transport to tissues and may contribute to earlier onset of fatigue and ischemic symptoms during submaximal exercise[22,23].

In athletic settings, elevated $PM_{2.5}$ concentrations have also been associated with reduced exercise efficiency and impaired performance outcomes, likely mediated by increased systemic inflammation, vascular dysfunction, and delayed post-exercise recovery[21,23].

## 8. Prevention Strategies and Practical Implications for Training in Polluted Environments

An increasing body of scientific evidence suggests that although regular physical activity confers substantial health benefits, exposure to air pollution during exercise may partially attenuate these positive effects [14]. Consequently, sports medicine literature has increasingly emphasized strategies aimed at minimizing health risks while optimizing training conditions.

### 8.1 Monitoring Air Quality and Training Planning

One of the primary preventive strategies involves real-time monitoring of air quality and adjusting training timing and location according to environmental conditions [14].

Diurnal variability in concentrations of particulate matter and ozone may significantly influence exposure levels during exercise. Peak ozone concentrations are typically observed during afternoon hours due to photochemical reactions driven by solar radiation [10]. In contrast, traffic-related pollutants such as $PM_{2.5}$ and nitrogen dioxide ($NO_2$) often reach their highest levels during morning and evening rush hours [10,24]. Appropriate scheduling of training sessions may therefore reduce inhaled pollutant dose without requiring cessation of physical activity.

In elite sport, selecting training routes away from major roadways is also recommended. Studies have demonstrated that even small increases in distance from traffic sources may result in substantial reductions in exposure to $PM_{2.5}$ and $NO_2$ [14,25].

Exercise intensity represents another key determinant of pollutant dose. Increased minute ventilation during high-intensity exercise substantially elevates inhaled pollutant load [13]. On days with elevated pollution levels, reducing training intensity or replacing high-intensity sessions with low-intensity or recovery-oriented exercise may mitigate oxidative stress and inflammatory responses[8,26].

The main preventive strategies for minimizing health risks during training in polluted environments are summarized in Table 1.

**Table 1.** Preventive strategies for minimizing exposure to air pollution during physical training.

| Strategy | Mechanism | Practical Application in Sport | Scientific Basis |
|---|---|---|---|
| **Monitoring air quality (AQI)** | Reduces exposure dose by avoiding peak pollution periods | Training in the morning instead of afternoon (high $O_3$), avoiding rush hours | WHO global air quality guidelines [9]; Giles LV, Koehle MS. The health effects of exercising in air pollution. [14] |
| **Selection of training location** | Increasing distance from emission sources lowers $PM_{2.5}$ and $NO_2$ concentration | Choosing parks, forests, routes away from major roads | WHO global air quality guidelines [9] |
| **Modification of training intensity** | Lower minute ventilation reduces pollutant inhalation | Replacing high-intensity intervals with low-intensity or recovery sessions on high-pollution days | Giles LV, Koehle MS. The health effects of exercising in air pollution. [14] |
| **Integration of environmental and physiological monitoring** | Individualized training adjustments based on exposure and physiological load | Combining AQI data with HR and ventilation monitoring | Al-Kindi SG, et al. Environmental determinants of cardiovascular disease: lessons learned from air pollution.. [22] |
| **Education of athletes and staff** | Improves risk awareness and decision-making | Training planning based on pollution forecast | WHO global air quality guidelines [9] |

### 8.2 Education of Athletes and Medical Staff

Education of athletes, coaches, and sports medicine personnel is a critical component of prevention strategies. Awareness of the potential impact of air pollution on health and performance enables more informed training decisions and improved load management [8].

In elite sport environments, environmental data are increasingly integrated with physiological monitoring parameters, such as heart rate and ventilatory responses, to individualize training recommendations [14]. Optimizing environmental conditions may influence not only athlete health but also training efficiency and competitive performance outcomes.

## 9. Discussion

The literature review presented in this study indicates that the impact of air pollution on athletes' health and physical performance is multifactorial and involves respiratory, cardiovascular, and systemic inflammatory mechanisms. Increased minute ventilation during exercise results in a greater inhaled dose of pollutants, distinguishing physically active individuals from the general population and partially modifying the benefit-risk balance associated with training in environments with reduced air quality [7,14].

### 9.1 Interpretation of the Impact of Air Pollution on Physical Performance

Evidence regarding performance-related parameters suggests that short-term exposure to ozone and particulate matter may lead to transient deterioration of pulmonary function, increased perceived exertion, and reduced time to exhaustion. These effects are mediated by oxidative stress, local inflammatory responses, and impaired oxygen transport [5].

However, findings across studies remain partially inconsistent. Some analyses report relatively small changes in $VO_2max$ and exercise economy, which may be explained by the high physiological adaptation level of endurance athletes. Systematic reviews emphasize that the magnitude of pollutant effects depends on pollutant type, exposure duration, exercise intensity, and individual susceptibility [14].

It should be noted that even small performance decrements, on the order of a few percent, may be meaningful in elite sport contexts. In recreational athletes, these effects may be less noticeable; however, chronic exposure may influence training adaptation by promoting inflammatory processes and prolonging post-exercise recovery [10,23].

### 9.2 Significance of Respiratory System Changes

The respiratory tract represents one of the primary target organs of environmental pollutants. Experimental studies have demonstrated transient reductions in spirometric parameters and increased bronchial hyperresponsiveness during exercise performed in polluted environments [7,12].

From a sports medicine perspective, the overlap between physiological exercise-induced inflammatory responses and pollution-induced airway inflammation is particularly important [16,17]. This interaction may increase susceptibility to exercise-induced asthma-like symptoms and recurrent respiratory infections, potentially impairing long-term training continuity [15,17].

### 9.3 Cardiovascular Consequences and Training Safety

Air pollution also affects cardiovascular function through endothelial dysfunction, increased sympathetic nervous system activity, and reduced heart rate variability. These mechanisms may contribute to elevated blood pressure and increased myocardial oxygen demand during exercise [8,20].

Although regular physical activity exerts strong cardioprotective effects [23], some authors suggest that high pollutant concentrations may attenuate these beneficial health effects [20]. Population-based analyses indicate that the health benefits of physical activity generally outweigh environmental risks, provided that exposure levels do not reach extremely high values [10,23].

### 9.4 Differences Between Athletes and the General Population

One of the key conclusions of the literature review is that athletes represent a population with a specific risk profile. On one hand, increased ventilation during exercise leads to a higher dose of inhaled pollutants. On the other hand, high cardiorespiratory fitness may partially protect against their adverse effects [14,26].

These differences may explain inconsistencies in epidemiological studies, where clear deterioration of athletes' health is not always observed despite environmental exposure. It is plausible that the net effect depends on the relationship between training intensity, pollution concentration, and exposure duration [26].

### 9.5 Practical Implications for Training and Sports Medicine

Based on the available literature, several practical recommendations can be identified. Monitoring air quality, adjusting training time and location, and avoiding high-intensity exercise during periods of elevated ozone or $PM_{2.5}$ concentrations may reduce adverse health effects [4,10].

From a clinical perspective, individualization of training recommendations appears particularly important for athletes with pre-existing respiratory diseases or cardiovascular risk factors. Incorporating environmental assessment into routine athlete care may represent an important preventive strategy[4,12].

### 9.6 Limitations of Current Research and Future Directions

Despite the growing number of publications, long-term prospective studies evaluating the impact of chronic pollution exposure on athletic career development and training adaptation remain limited. Many studies rely on laboratory models or short-term exposure assessments that may not fully reflect real training conditions [26].

Future research should consider differences between sports disciplines, training status, and sex, as well as interactions between air pollution and other environmental factors such as temperature and humidity[26].

### 9.7 Summary of Discussion

Overall, scientific evidence indicates that air pollution may negatively influence both health and athletic performance through respiratory, cardiovascular, and systemic inflammatory mechanisms. At the same time, regular physical activity remains one of the most important protective factors for health, and optimization of environmental training conditions plays a crucial role in risk mitigation. Current knowledge highlights the need for further research focused on athletic populations to better define safe exposure thresholds and develop precise recommendations for sports practice.

## 10. Conclusions

Based on the conducted literature review, it can be concluded that air pollution represents a significant and often underestimated environmental factor affecting both health and physical performance in athletes. The increase in minute ventilation during exercise leads to greater exposure to particulate matter and gaseous pollutants, which may initiate inflammatory processes and oxidative stress within the respiratory and cardiovascular systems.

The analysis of scientific evidence indicates that short-term exposure to air pollution may result in transient impairment of lung function, increased perceived exertion, and reduced exercise tolerance. In contrast, long-term exposure is associated with the risk of chronic airway alterations, vascular endothelial dysfunction, and increased cardiovascular burden.

Although a high level of physical fitness may partially attenuate the adverse effects of pollutant exposure, it does not completely eliminate health risks. From a sports medicine perspective, it is particularly important to recognize that symptoms related to pollution exposure—such as cough, dyspnea, or decreased performance—may be misinterpreted as signs of training overload.

Therefore, incorporating air quality assessment into the planning of training sessions and competitions appears to be an important component of preventive health strategies. Monitoring environmental indicators, modifying exercise intensity during periods of elevated pollution, and educating athletes may reduce potential adverse health consequences.

At the same time, available evidence suggests that the benefits of regular physical activity generally outweigh the risks associated with moderate exposure to polluted air. However, optimizing training conditions and individualizing recommendations according to pollution levels and the athlete’s health status remain essential.

Despite the growing number of publications in this field, there is still a need for long-term prospective studies evaluating the impact of chronic exposure on athletic career progression, training adaptations, and the risk of cardiovascular and respiratory diseases. Future research should also consider differences between sports disciplines, sex, and training status, which may enable the development of more precise recommendations for sports and clinical practice.

The exact dose–response relationship between air pollution exposure and exercise-related physiological adaptation remains incompletely defined.

No conflicts of interest to declare.

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