QUALITY IN SPORT

eISSN 2450-3118 · Open Access · Peer-reviewed

Vol. 55 (2026) · Article 72034 · Published 16 May 2026

apcz.umk.pl/QS · Nicolaus Copernicus University in Toruń

Cite as: **Korzeniowska A, Szafraniec A, Chmiela M. Double Jeopardy: The Cardiovascular Exposome of Athletes in Heat and Pollution: A Mini Review. Quality in Sport. 2026;56:72034. https://doi.org/10.12775/QS.2026.56.72034**

ARTICLE TIMELINE

Received: 18.05.2026 **Revised:** 05.05.2026

Accepted: 19.05.2026 **Published:** 30.05.2026

INDEXING & EVALUATION

MEiN points: 20 **Unique ID:** 201398

Disciplines: Medical Sciences; Health Sciences

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Medical Sciences; Health Sciences.

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Nauki medyczne; Nauki o zdrowiu. © The Authors 2026.

OPEN ACCESS · CC BY-NC-SA 4.0 This article is published with open access under the License Open Journal Systems of Nicolaus Copernicus University in Toruń, Poland, and is distributed under the terms of the Creative Commons Attribution Non-commercial Share Alike License (http://creativecommons.org/licenses/by-nc-sa/4.0/), which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the work is properly cited. The authors declare no conflict of interest regarding the publication of this paper.

MINI REVIEW

Double Jeopardy: The Cardiovascular Exposome of Athletes in Heat and Pollution

a mini review

HIGHLIGHTS

- Heat and air pollution interact synergistically to create a cardiovascular bottleneck in training athletes.
- Thermal vasodilation accelerates translocation of ultrafine particles, compounding myocardial oxygen supply-demand mismatch.
- Heart rate variability (HRV) declines acutely with PM2.5 and NO2 exposure, reflecting autonomic imbalance.
- Extended heat acclimation (≥10 days) is mandatory for complete renal protection and full cardiovascular adaptation.
- Multi-sensor wearables and continuous longitudinal monitoring are the new clinical standard for athletes in hostile environments.

AUTHORS & AFFILIATIONS

Anna Korzeniowska [AK]
ORCID: https://orcid.org/0009-0003-6288-3400
E-mail: korzeniowskaa6@gmail.com
Wrocław University Hospital, Borowska 213, 50-556 Wrocław, Poland

Artur Szafraniec [AS]
ORCID: https://orcid.org/0000-0002-9991-2039
E-mail: aszafraniec15@gmail.com
Lower Silesian Centre for Oncology, Pulmonology and Haematology, plac Hirszfelda 12, 53-413 Wrocław, Poland

Mateusz Chmiela [MC]
ORCID: https://orcid.org/0009-0008-2317-0594
E-mail: mchmielusz@gmail.com
Health Care Center in Olawa, K.K. Baczynskiego 1, 55-200 Olawa, Poland

CORRESPONDING AUTHOR **Anna Korzeniowska –** korzeniowskaa6@gmail.com

ABSTRACT

BACKGROUND: Ambient heat and air pollution form a “hostile environment” that triggers a severe cardiovascular bottleneck, forcing a direct conflict between thermoregulatory skin blood flow and working muscles while inhaled pollutants drive systemic inflammation.

AIM: To synthesize the compound cardiovascular risks of this dual-stressor exposome and establish a practical clinical framework to safeguard athletes.

MATERIAL AND METHODS: A narrative synthesis of recent literature was performed, evaluating physiological, multi-omics, and epidemiological data on the cardiorespiratory impacts of exercising under thermal and pollutant stress.

RESULTS: Heat and pollution interact synergistically to degrade athletic output. Thermal vasodilation accelerates the translocation of ultrafine particles into the bloodstream, compounding myocardial oxygen supply-demand mismatches, decreasing heart rate variability (HRV), and inducing a pro-thrombotic state. Acclimation protocols might become a crucial strategy in hostile training.

CONCLUSIONS: The compound strain of heat and pollution elevates cardiac risk and shortens time to exhaustion. Clinicians must transition to continuous longitudinal monitoring, mandate acclimation protocols, and engage in environmental health advocacy.

KEYWORDS Cardiovascular exposome; Heat acclimation; Particulate matter (PM2.5); Thermoregulation; Wearable sensors; Environmental cardiology.

PLAIN LANGUAGE SUMMARY

When athletes train in hot, polluted air, their hearts face two simultaneous threats. Heat forces the body to divert blood to the skin for cooling, while air pollution — particularly fine particles (PM2.5) and ozone — inflames blood vessels and disrupts the heart's rhythm. Together, these stressors create a "double hit" that raises the risk of heart attack, arrhythmia, and heat stroke far beyond what either factor causes alone. Practical solutions include training in the early morning, staying at least 100 metres from busy roads, completing a 10–14 day heat acclimation programme before major events, and using wearable sensors to monitor heart rate variability and core temperature in real time.

TABLE OF CONTENTS

Section titles below are listed with their page numbers in the printed and PDF layout.

Abstract

Plain Language Summary

Table of Contents

1. Introduction

2. Methodology

3. Acute Physiological Conflict: Heat & Hemodynamics

4. Pollution & Autonomic Balance

5. The Combined Effect of Heat and Air Pollution During Training

6. Practical Mitigation for the Clinician
7. Conclusion
8. Disclosure
References

1. INTRODUCTION

The "hostile environment" — a composite stressor characterized by the intersection of ambient heat and air pollution — represents an increasingly recognized challenge for modern athletes. While exercise capacity is traditionally viewed through the lens of metabolic fatigue, in hostile environments, it is governed by a cardiovascular bottleneck. This occurs when the mandatory demand for thermoregulatory skin blood flow competes with the requirements of the working muscles, all while systemic inflammation from inhaled pollutants impairs the heart's autonomic and vascular efficiency.

Whilst both stressors have been studied independently, their specific interaction during physical exertion remains incompletely characterized in current literature. Real-world data confirms that heat and air pollution independently impair athletic performance, yet evidence suggests their combined impact may be additive or even synergistic in nature. [1] Large-scale longitudinal analysis, such as that conducted on the Great North Run half marathon from 2006 to 2019, demonstrates that increases in temperature, heat index, and ozone concentrations are significantly detrimental to runner performance. [1,2]

The physiological impact of this composite stressor is systemic, affecting respiratory, cardiovascular, and hematological functions. Exercise in polluted air significantly decreases peak expiratory flow (effect size −0.238), while ozone exposure during moderate-to-vigorous activity carries high-certainty evidence for adverse pulmonary effects. [3,4] Meta-analyses confirm that FEF25-75 declines significantly ($g = -0.323$) alongside smaller reductions in FEV1 ($g = -0.183$) and FVC ($g = -0.131$) when exercising in polluted environments. [5] Cardiovascularly, consistent rises in systolic blood pressure ($g = 0.279$) have been observed, and while heat exposure independently increases cardiovascular risk, these risks are amplified when combined with fine particulate air pollution. [5,6]

While modeling suggests that for healthy individuals, the benefits of exercise generally outweigh pollution risks — except in extremely polluted cities (annual PM2.5 > 100 μg/m3) — this calculus may shift under combined heat-pollution exposure. [7] Currently, organizations like the Canadian Academy of Sport and Exercise Medicine and the IOC emphasize mitigation strategies such as air quality education, monitoring, acclimation protocols, and the use of N95 masks. [8,9]

2. METHODOLOGY

Framed within the field of environmental cardiology, this mini-review employs a narrative synthesis approach to evaluate how ambient heat and particulate matter (PM2.5) concurrently disrupt the cardiovascular exposome of athletes. A comprehensive literature search was conducted across PubMed, Web of Science, and Google Scholar for peer-reviewed studies, systematic reviews, and consensus statements published between 2019 and 2026. The search strategy integrated terms targeting environmental stress and thermoregulation with specific cardiorespiratory pathophysiologies and clinical interventions, notably structured heat acclimation and real-time tracking via wearable sensors. Literature selection prioritized high-certainty randomized controlled trials, longitudinal cohorts, and official guidelines from major global medical bodies, including the AHA, ESC, and IOC.

3. ACUTE PHYSIOLOGICAL CONFLICT: HEAT & HEMODYNAMICS

The cardiovascular response to physical exertion in extreme thermal environments presents a profound conflict in resource allocation. When an athlete trains in extreme heat, the body must dissipate core heat primarily through peripheral vasodilation, which drastically increases cutaneous blood flow. This massive redirection of blood away from the central circulation to the skin lowers systemic vascular resistance and reduces available plasma volume. To maintain blood pressure and preserve cardiac output in the face of a diminished stroke volume, the body relies on a compensatory increase in heart rate, known as tachycardia. [10,11] On average, for every 1°C increase in core body temperature, the heart rate rises by an average of 26 bpm, while systolic blood pressure can drop by 8 mmHg. [10]

This rapid hemodynamic shift creates a severe myocardial supply-demand mismatch. The combination of heat-induced tachycardia and reduced stroke volume elevates myocardial oxygen demand. [10] Simultaneously, extreme tachycardia shortens the diastolic filling time of the cardiac cycles. [11] Because the coronary arteries primarily perfuse the myocardium during diastole, operating at this physiological ceiling can compromise myocardial perfusion during peak exertion, substantially increasing the risk of ischemia or arrhythmias in susceptible individuals. [12]

This hemodynamic conflict extends deep into internal organs. During exercise, renal blood flow is reduced by 25% to 50% compared to resting levels. This effect is heavily intensified in hot environments as skin blood flow and sweating rates climb to facilitate cooling. [13] This triggers a profound neurohumoral response characterized by heightened sympathetic outflow, catecholamine release, and arginine vasopressin (AVP) secretion. Five days of heat acclimation (HA) can successfully reduce resting heart rate by 8±5 bpm and expand plasma volume by 7.3%, reflecting improved stroke volume and cardiac function. [13]

Vulnerability to this acute conflict varies across lifespans and environments. Children possess an immature thermoregulatory capacity that accelerates core temperature rise. In pediatric cohorts, short-term thermal stress provokes rapid electrophysiological responses, triggering tachycardia and inducing functional cardiovascular alterations — such as a decreased Ejection Fraction (EF) and reduced E/A ratio — well before permanent structural remodeling occurs. [15] Furthermore, when heat is paired with ambient gaseous pollutants like ozone (O3) and nitrogen dioxide (NO2), cardiopulmonary function is restricted further during maximal exercise in well-trained athletes. [16]

4. POLLUTION & AUTONOMIC BALANCE

Exertion in urban environments exposes athletes to airborne toxins that acutely disrupt the autonomic nervous system (ANS) and cardiac function. [11] Short-term exposure to Fine Particulate Matter (PM2.5) and NO2 triggers an abrupt autonomic shift characterized by an increase in resting and submaximal heart rates alongside a sharp decline in heart rate variability (HRV) indices. Specifically, acute reductions are observed in the standard deviation of normal-to-normal intervals (SDNN) and the percentage of successive RR intervals differing by more than 50 ms (pNN50). This shift is primarily driven by immediate sympathetic stimulation, which prompts the release of adrenaline and norepinephrine to directly elevate heart rate. [18]

Beyond direct neural disruption, inhaled pollutants cause widespread systemic damage. PM2.5 stands as the leading environmental risk factor for cardiovascular disease and mortality. Upon inhalation, these small pollutants cross the lung-blood barrier to enter the systemic circulation directly. In the pulmonary and vascular tissues, pollutants trigger the production of reactive oxygen species (ROS) and launch a robust inflammatory cascade, elevating systemic markers such as interleukin-6 (IL-6) and C-reactive protein (CRP). The combination of systemic inflammation and oxidative stress induces profound endothelial dysfunction, increases blood viscosity, and enhances platelet activation, leaving the athlete in a highly vulnerable, pro-thrombotic state. [10,17,18]

Athletes are uniquely vulnerable to this pathophysiology due to the mechanics of exercise hyperpnea, often referred to as the “ventilation-perfusion” dose effect. During intense exercise, minute ventilation spikes from a resting rate of roughly 6 L/min to over 100–200 L/min. This massive volume of air is typically sustained via mouth breathing, which entirely bypasses the nasal filtration system. Consequently, a significantly higher load of PM2.5 and ultrafine particles penetrates deep into the alveolar regions and enters the systemic circulation. [11,16,17]

Recent multi-omics data underscores these findings, showing that PM2.5 exposure is significantly tied to a decline in both cardiac systolic functions (such as EF and fractional

shortening) and diastolic functions (E/A and e' ratios) via pathways of nervous system dysregulation and oxidative stress. On a molecular level, lipidomic profiling identifies changes in triacylglycerides (TAGs) and hexosylceramides (HexCers) as key mediators. Concurrently, long-term pollution and humidity variations downregulate Myosin Light Chain 3 (MYL3), predisposing the heart to apical aneurysms and sudden cardiac death. [15]

5. THE COMBINED EFFECT OF HEAT AND AIR POLLUTION DURING TRAINING

When extreme heat and air pollution coincide, they do not act as isolated stressors; rather, they exhibit a hazardous synergistic relationship that amplifies cardiovascular risk and severely degrades athletic output. Environmental extremes drastically modify the body's normal physiological response to training, lowering exercise capacity and compounding cardiovascular strain through the triple threat of high core temperatures, progressive dehydration, and dropping stroke volumes. Under intense heat, the required physical labor or sport-specific exertion heavily intensifies tachycardia, exacerbating the myocardial oxygen supply-demand mismatch. When high humidity or prolonged exertion is added to the mix, the body's native thermoregulatory mechanisms are easily overwhelmed, paving the way for collapse or heat stroke. [10,17]

The coexistence of high temperatures directly worsens the pollution profile. Elevated ambient temperatures accelerate the atmospheric formation of ground-level ozone and aggravate wildfire-related PM2.5, effectively increasing the concentration and "dose" of pollutants an individual receives. [10,16] At the same time, co-exposure to nonoptimal temperatures and particulate matter heightens cardiovascular risk by acting on shared pathophysiological pathways: both independently drive oxidative stress, systemic inflammation, neurohormonal activation, and endothelial dysfunction. Heat-induced hypovolemia and hemoconcentration work in tandem with pollution-induced platelet activation to create a hypercoagulable, highly pro-thrombotic state, multiplying the immediate risk of stroke or myocardial infarction. [10]

Crucially, the cardiovascular response to heat alters the body's internal handling of toxins through facilitated translocation. To lose heat, the body undergoes profound systemic vasodilation, accelerating the movement of blood through the peripheral and pulmonary capillaries. [11,17] When an athlete couples this rapid circulatory state with the massive ventilation rates demanded by training, it alters the permeability of the alveolar-capillary membrane. This allows ultrafine particles and gaseous pollutants to translocate from the lungs into the systemic bloodstream much more rapidly, profoundly worsening the systemic inflammatory and oxidative stress response. [11]

This "double hit" on the cardiorespiratory system imposes an intense compound strain. [15] The interaction between thermal and pollutant stress markedly elevates an athlete's perceived

rate of exertion (RPE) and significantly shortens the time to exhaustion, effectively “lowering the ceiling” of achievable athletic performance. [11] Aerobic fitness and endurance drop significantly as ozone concentrations rise. [16] Furthermore, these interactive effects are highly intensity-dependent; higher training intensities and longer exposure durations accelerate the environment-cardiovascular axis, driving accelerated, adverse cardiac remodeling over time. [15]

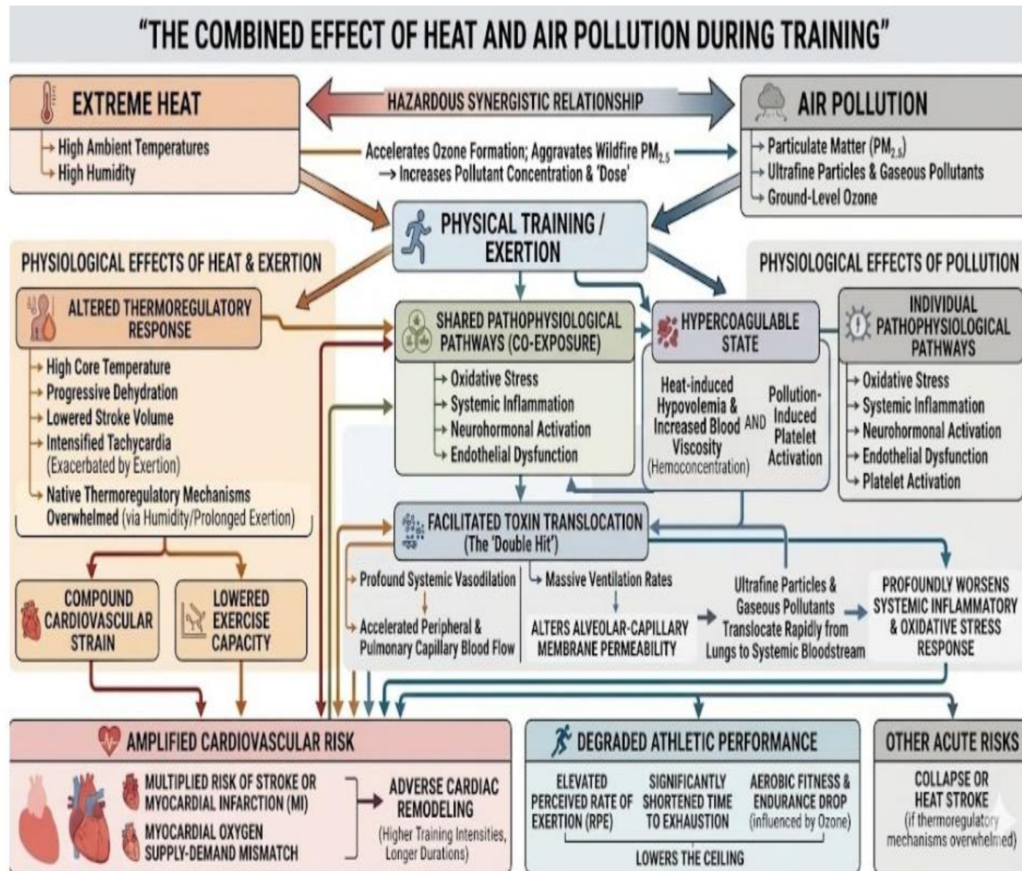

Figure 1. The combined effect of heat and air pollution during training.

6. PRACTICAL MITIGATION FOR THE CLINICIAN

6.1. Advanced Screening & Continuous Monitoring

To protect athletes training in hostile environments, clinicians must transition from traditional, episodic laboratory testing to continuous, longitudinal screening strategies. [19] Heart rate variability (HRV) serves as a vital, non-invasive tool to gauge cardiac risk and mortality. Reviewing morning resting HRV can reveal a drop in parasympathetic tone, signaling “over-reaching” during training camps, while serial HRV and ambulatory ECG monitoring can capture early signs of autonomic imbalance or pollution-triggered arrhythmias in high-AQI zones. [11,12,14,18] Because functional cardiac changes manifest rapidly before structural

remodeling takes place, clinicians should prioritize echocardiographic tracking of diastolic function (such as EF and E/A ratios) and ion-channel stability for athletes exposed to prolonged environmental stress. [15]

Integrating modern wearables offers a non-invasive paradigm shift for real-time tracking. Moving away from surface skin sensors, ear-worn core temperature sensors yield highly accurate measurements of internal cardiac and thermal strain under hostile conditions. Clinicians can leverage specific biofluids to monitor real-time physiological shifts: sweat wearable microfluidic sensors track sodium, lactate, cortisol, and sweat osmolality to prevent dehydration, metabolic exhaustion, and heat-related illness; saliva and tear cortisol sensors track hypothalamic-pituitary-adrenal (HPA) axis activity; and urine sensors catch early markers of kidney stress like proteinuria. [19]

The ultimate clinical goal of these multi-omics and multi-sensor wearable systems is the implementation of predictive warning systems. Instead of merely logging stress, these devices are designed to alert a coach or clinician that an athlete's performance and physiological thresholds are about to drop, allowing for immediate, pre-emptive cooling, forced hydration, or rest. [20] For comprehensive clinical screening, emerging molecular biomarkers offer distinct advantages: utilizing Neutrophil Gelatinase-Associated Lipocalin (NGAL) and Kidney Injury Molecule-1 (KIM-1) provides a highly sensitive look at early renal tubular stress. [13]

In terms of functional diagnostics, standard Heat Stress Testing (HST) — such as a 45-minute cycling test at 32°C and 70% relative humidity — is highly recommended to evaluate an athlete's physiological and perceptual response to heat. [11,13] For elite endurance athletes, isothermic (controlled hyperthermia) protocols tracking real-time core temperature are superior to fixed-intensity tests for ensuring the athlete safely reaches and sustains the necessary adaptation threshold of 38.5°C without excessive strain. [12,21]

6.2. Benefit-Risk Dynamics & Exertional Thresholds

The clinician must recognize that the "Benefit-Risk" threshold of exercise is highly dynamic and heavily modified by the surrounding exposome. [11,17] For air pollution, there may not be a completely "safe" baseline threshold for vulnerable individuals; low ambient concentrations well below current regulatory guidelines can still impair autonomic balance, and ozone has been shown to degrade athletic performance even below the standard WHO guideline of ~81 μg/m3. [16]

When analyzing temperature, the cardiovascular risk follows a distinct U-shaped or J-shaped curve, where the nadir represents the "optimal temperature" carrying the lowest statistical risk of cardiac events for a given geographic region. [10] For high-intensity athletic training, a critical tipping point occurs as the Air Quality Index (AQI) exceeds 150; at this juncture, the

environmental risk completely outweighs the performance or health benefits of the session, dictating that training be moved indoors to an environment with HEPA filtration, or that the intensity be drastically minimized. [11]

In highly polluted zones where PM2.5 exceeds 100 μg/m3, the health benefits of exercise peak and begin to decline after a mere 15 minutes of exposure. After approximately 75 minutes of moderate-to-high intensity activity in these severely polluted conditions, the session reaches a "break-even point" where the environmental risks completely surpass any exercise-induced cardiovascular benefits. [11] Similarly, an exertional wet-bulb globe temperature (WBGT) threshold of 31–32°C represents a point of diminishing returns, where continuing to train spikes the risk of acute, heat-induced myocardial strain unless a rigorous 10–14 day acclimation protocol has been completed. [12,21]

6.3. Subgroup Vulnerabilities

When prescribing exercise or clearing athletes, clinicians must evaluate several risk multipliers. Older smokers exhibit distinct, highly attenuated or altered autonomic responses to pollution, showing smaller absolute physiological changes but carrying elevated baseline risk. [18] Individuals from low-income households face significantly higher susceptibility to environmental cardiac remodeling due to higher baseline exposures. [18] Stricter screening must be applied to those with pre-existing elevated blood pressure, diabetes, or cardiovascular diseases (CVD). [18] Individuals taking beta-blockers show significantly stronger associations between NO2 exposure, increased heart rate, and decreased RMSSD. Clinicians must closely review electronic health records (EHR) and toolkits to identify medications that impair thermoregulatory capacity, such as antihypertensives or diuretics, and carefully titrate them prior to forecasted heat waves. [10,18]

Gender-specific dynamics must also be considered: air quality thresholds must be tailored specifically for female athletes. Due to physiological differences — including smaller absolute lung volumes and distinct hormonal profiles that modulate pollutant impact — their performance and health are affected differently. Furthermore, an elevated body fat percentage acts as a performance and thermal risk multiplier during environmental stress, compromising output further in high-pollution zones. [16]

6.4. Structured Interventions & Policy

To successfully adapt athletes to hostile environments, structured Heat Acclimation (HA) protocols are paramount, though clinicians must avoid the "training status" paradox. Highly endurance-trained athletes are often "partially acclimated" purely from their high-volume training, yet they actually require a "higher thermal impulse" (longer or hotter exposures) to trigger further cardiovascular adaptations compared to recreationally active individuals. [12,14,21]

Importantly, short-term HA protocols yield incomplete protection. A 5-day HA protocol successfully improves basic cardiovascular and thermoregulatory responses — reducing resting heart rate by 8±5 bpm and expanding plasma volume by 7.3%. However, data reveals that 5 days of HA is entirely insufficient to reduce renal tubular stress or fluid-regulatory strain. Clinicians must understand that early cardiovascular improvements do not equal renal protection; consequently, extended HA protocols lasting 10 to 14 days (≥10 days) are mandatory to induce a complete, fully protective heat-adapted phenotype. [13]

On a daily operational level, clinicians and coaches should utilize specific environmental behaviors: strategic scheduling using predictive ambient air quality and meteorological models to select optimal locations and time periods [11,13,16]; diurnal planning to avoid peak afternoon ozone hours and peak morning traffic emissions, prioritizing the cooler, cleaner early morning hours [11]; spatial separation by moving training sessions at least 50–100 metres away from major roadways to exponentially reduce direct exposure to particulate matter [11,16]; and dietary countermeasures such as recommending a high daily intake of antioxidants, specifically Vitamins C and E, to offer a modest protective effect by helping to scavenge free radicals and oxidative stress triggered by particulate matter exposure. [11]

Finally, leading global cardiovascular bodies (including the ESC, ACC, AHA, and WHF) have united to officially recognize the Environmental Risk Factor (ERF) as a primary, preventable driver of global CVD. They urge the medical community to shift to a comprehensive, exposome-based strategy that accounts for the compounding, cumulative risks of air, water, soil, noise, light, and climate pollution acting together. Clinicians are called to move beyond standard risk factors to actively screen patients for environmental exposures. [17]

7. CONCLUSION

The intersection of extreme ambient heat and air pollution creates a hazardous, synergistic “hostile environment” that imposes a severe cardiovascular bottleneck and elevates systemic risk for training athletes. This dual-stressor exposome triggers an acute physiological conflict where accelerated pollutant translocation, autonomic imbalance, and a highly pro-thrombotic state collide with intense myocardial and renal tubular strain. Safely managing athletes within this narrow safety window requires clinicians to shift from episodic laboratory testing toward continuous, longitudinal monitoring using multi-sensor wearables and advanced molecular screening. Furthermore, implementing extended heat acclimation protocols lasting at least 10 to 14 days is mandatory to ensure complete renal protection and full cardiovascular adaptation. Ultimately, sports medicine clinicians must adopt a comprehensive, exposome-based strategy and actively step into advocacy roles to demand stricter systemic environmental regulations to safeguard athletes worldwide.

8. DISCLOSURE

8.1. Author Contributions

Conceptualization: Anna Korzeniowska. Methodology: Anna Korzeniowska, Artur Szafraniec, Mateusz Chmiela. Software: Not applicable. Check (Validation): Artur Szafraniec, Mateusz Chmiela. Formal analysis: Artur Szafraniec, Mateusz Chmiela. Investigation: Anna Korzeniowska, Artur Szafraniec, Mateusz Chmiela. Resources: Anna Korzeniowska, Artur Szafraniec, Mateusz Chmiela. Data curation: Anna Korzeniowska, Artur Szafraniec, Mateusz Chmiela. Writing – rough preparation: Anna Korzeniowska, Artur Szafraniec, Mateusz Chmiela. Writing – review and editing: Anna Korzeniowska, Artur Szafraniec. Visualization: Anna Korzeniowska. Supervision: Anna Korzeniowska. Project administration: Anna Korzeniowska. All authors have read and agreed with the published version of the manuscript.

8.2. Funding

This study has not received any external funding.

8.3. Institutional Review Board Statement

Not applicable.

8.4. Informed Consent Statement

Not applicable.

8.5. Data Availability Statement

Not applicable.

8.6. Acknowledgements

Not applicable.

8.7. Conflict of Interest

The authors declare that there are no financial or personal relationships with other people or organizations that could inappropriately influence (bias) their work.

8.8. CRediT Author Contributions (taxonomy)

Mapped to the CRediT (Contributor Roles Taxonomy, NISO Z39.104-2022). Author initials: AK=Anna Korzeniowska; AS=Artur Szafraniec; MC=Mateusz Chmiela.

- Conceptualization: AK
- Methodology: AK, AS, MC
- Software: Not applicable
- Validation (Check): AS, MC

• Formal analysis: AS, MC

• Investigation: AK, AS, MC

• Resources: AK, AS, MC

• Data curation: AK, AS, MC

• Writing – original draft: AK, AS, MC

• Writing – review & editing: AK, AS

• Visualization: AK

• Supervision: AK

• Project administration: AK

• Funding acquisition: Not applicable

Declaration of the use of generative AI and AI-assisted technologies in the writing process

In preparing this work, the authors used AI tools (ChatGPT, Monica AI) for the purpose of language editing, improving readability, and text formatting. After using these tools, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

REFERENCES

[1] Hodgson JR, Chapman L, Pope FD. Amateur runners more influenced than elite runners by temperature and air pollution during the UK's Great North Run half marathon. Sci Total Environ. 2022;842:156825. https://doi.org/10.1016/j.scitotenv.2022.156825

[2] Lee HY, Kim HJ, Kim HJ, Na G, Jang Y, Kim SH, et al. The impact of ambient air pollution on lung function and respiratory symptoms in elite athletes. Sci Total Environ. 2023;855:158862. https://doi.org/10.1016/j.scitotenv.2022.158862

[3] Qin F, Yang Y, Wang ST, Dong YN, Xu MX, Wang ZW, et al. Exercise and air pollutants exposure: a systematic review and meta-analysis. Life Sci. 2019;218:153-164. https://doi.org/10.1016/j.lfs.2018.12.036

[4] Hung A, Nelson H, Koehle MS. The acute effects of exercising in air pollution: a systematic review of randomized controlled trials. Sports Med. 2022;52(1):139-164. https://doi.org/10.1007/s40279-021-01544-4

[5] Jiang S, Su Y, Li H, Cao Y, Zhang G, Dzhambov AM. Effects of physical activity in polluted air on physiological health in healthy populations: a systematic review and meta-analysis of controlled trials. Ecotoxicol Environ Saf. 2026;315:120098. https://doi.org/10.1016/j.ecoenv.2026.120098

[6] Bell ML, Gasparrini A, Benjamin GC. Climate change, extreme heat, and health. N Engl J Med. 2024;390(19):1793-1801. https://doi.org/10.1056/NEJMra2210769

[7] Rajagopalan S, Brauer M, Bhatnagar A, Bhatt DL, Brook JR, Huang W, et al. Personal-level protective actions against particulate matter air pollution exposure: a scientific statement from the American Heart Association. Circulation. 2020;142(23):e411-e431. https://doi.org/10.1161/CIR.0000000000000931

[8] Hung A, Koch S, Bougault V, Gee CM, Bertuzzi R, Elmore M, et al. Personal strategies to mitigate the effects of air pollution exposure during sport and exercise. Br J Sports Med. 2023;57(4):193-202. https://doi.org/10.1136/bjsports-2022-106161

[9] Bougault V, Carlsten C, Adami PE, Anderson S, Bartsch P, Bonini M, et al. Air quality, respiratory health and performance in athletes: a summary of the IOC consensus subgroup narrative review. Br J Sports Med. 2025;59(7):480-490. https://doi.org/10.1136/bjsports-2024-109145

[10] Hanneman K, Alahmad B, Ghosh A, Khatana SAM, Huang M, Liu J, et al. Nonoptimal temperature and cardiovascular health: a scientific statement from the American Heart Association. Circulation. 2026;153(16):e1130-e1150. https://doi.org/10.1161/CIR.0000000000001419

[11] Sobolewska E, Markowski D, Baranowska M, Dziaman J, Kretschmer V, Daniszewski W, et al. Impact of air pollution on athletic performance and health: a comprehensive review. Int J Innov Technol Soc Sci. 2026;1(2(50)):5119. https://doi.org/10.31435/ijitss.2(50).2026.5119

[12] McDonald P, Brown HA, Topham TH, Kelly MK, Jardine WT, Carr A, et al. Influence of exercise heat acclimation protocol characteristics on adaptation kinetics: a quantitative review with Bayesian meta-regressions. Compr Physiol. 2025;15(3):e70017. https://doi.org/10.1002/cph4.70017

[13] Snape D, Wainwright B, Parsons IT, Stacey MJ, Woods DR, O'Hara J. Five days of heat acclimation improves cardiovascular and thermoregulatory responses without altering renal stress biomarkers in endurance athletes. bioRxiv. 2026. https://doi.org/10.64898/2026.03.06.710014

[14] Periard JD, Eijsvogels TMH, Daanen HAM. Exercise under heat stress: thermoregulation, hydration, performance implications, and mitigation strategies. Physiol Rev. 2021;101(4):1873-1979. https://doi.org/10.1152/physrev.00038.2020

[15] Zhang D, Liang F, Li Q, Chen J, An X, Luo S, et al. Air pollution, meteorological factors, and cardiac remodelling in children: a multi-omics cohort study. Eur Heart J. 2026;47(15):1829-1843. https://doi.org/10.1093/eurheartj/ehaf1086

[16] Xing W, Wang Y, Xie Y, Zheng W. Ozone pollution impairs athletic performance in female football players: a gender-specific analysis. Atmosphere. 2025;16(7):834. https://doi.org/10.3390/atmos16070834

[17] Munzel T, Luscher T, Kramer CM, Churchwell K, Mbakwem A, Rajagopalan S, et al. Environmental stressors and cardiovascular health: acting locally for global impact in a changing world. Circulation. 2026. https://doi.org/10.1161/CIRCULATIONAHA.125.079034

[18] Li Y, Breitner-Busch S, Cascio WE, Zhang S, Wolf K, Ruckert-Eheberg IM, et al. Short-term association between ambient air pollution and heart rate variability: results from the population-based

KORA S4 and FF4 studies. Part Fibre Toxicol. 2025;22(1):26. https://doi.org/10.1186/s12989-025-00645-6

[19] Gulgosteren E, Agrali Ermis S, Algin Toros A, Toros T, Serin E, Sekeroglu MO, et al. Sweat, tears, and beyond: advanced wearable sensors for personalized health and athletic performance. Front Bioeng Biotechnol. 2025;13:1684674. https://doi.org/10.3389/fbioe.2025.1684674

[20] Flintoff JM, Pattinson C, Ahamed S, Ali S, Bagley A, Broszczak D, et al. Predictive biomarkers of performance under stress: a two-phase study protocol to develop a wearable monitoring system. BMJ Open Sport Exerc Med. 2025;11(1):e002410. https://doi.org/10.1136/bmjsem-2024-002410

[21] Moss JN, Naughton MR, Mackenzie RWA, Trangmar SJ, Reeve TC, Tyler CJ. The effects of isothermic heat acclimation on simple and complex cognitive performance in the heat. J Sports Sci. 2026. https://doi.org/10.1080/02640414.2026.2619323

• • •

Quality in Sport · Nicolaus Copernicus University in Toruń · apcz.umk.pl/QS