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Association of exposure to air pollutants and risk of mortality among people living with HIV: a systematic review

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Abstract

Background People living with HIV (PLWH) are more vulnerable to infectious and non-infectious comorbidities due to chronic inflammation and immune dysfunction. Air pollution is a major global health risk, contributing to millions of deaths annually, primarily from cardiovascular and respiratory diseases. However, the link between air pollution and mortality risk in PLWH is underexplored. This systematic review assesses the association between exposure to pollutants such as particulate matter (PM), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO) and mortality risk in PLWH.

Methods A systematic search of PubMed, Web of Science, and Embase was conducted for studies published up to August 2024. Eligibility criteria included cohort, case-control, and cross-sectional studies assessing air pollution exposure and mortality in PLWH. Nested-Knowledge software was used for screening and data extraction. The Newcastle-Ottawa Scale was applied for quality assessment. A narrative approach and tabular summarization were used for data synthesis and presentation.

Results Nine studies, mostly from China, demonstrated a significant association between long-term exposure to PM^1, $PM^{2.5}$, and PM^{10} and increased risks of AIDS-related and all-cause mortality in PLWH. Hazard ratios for mortality increased by 2.38–5.13% per unit increase in PM concentrations, with older adults (> 60), females, and those with lower CD4 counts (< 500 cells/μL) being more vulnerable. Short-term exposure to ozone and sulfur dioxide also increased mortality risks, particularly during the warm season and in older populations. Specific pollutants like ammonium ($NH4^+$) and sulfate ($SO4^{2-}$) had the strongest links to elevated mortality.

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Conclusion Air pollution, especially fine particulate matter and ozone, is associated with a higher risk of mortality in PLWH. Targeted interventions to reduce pollution exposure in vulnerable subgroups are crucial. Further research is needed to confirm these findings in diverse regions and develop effective mitigation strategies.

Keywords Air pollution, People living with HIV, Mortality, Particulate matter, Nitrogen dioxide

Introduction

Human immunodeficiency virus (HIV) remains a significant global public health concern despite substantial advancements in treatment, care, and prevention strategies [1]. While antiretroviral therapy (ART) has dramatically reduced the morbidity and mortality associated with HIV, enabling many to live longer, healthier lives, people living with HIV (PLWH) continue to face unique health challenges [2]. Among these challenges is an increased vulnerability to both infectious and non-infectious comorbidities, including cardiovascular diseases, respiratory infections, and various cancers [3–6]. This heightened susceptibility to comorbid conditions can be attributed to the chronic inflammation and immune dysregulation caused by the virus, as well as the long-term effects of ART.

Concurrently, air pollution has emerged as a leading environmental risk factor for global morbidity and mortality [7, 8]. The World Health Organization (WHO) estimates that exposure to ambient air pollution is responsible for approximately 6.7 million deaths annually, largely due to cardiovascular and respiratory diseases, as well as cancers [8]. The health impacts of air pollution are wide-ranging and include increased risks of asthma, chronic obstructive pulmonary disease (COPD), ischemic heart disease, stroke, and lung cancer [9]. The primary air pollutants of concern include particulate matter (PM), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO), each of which can have harmful effects on human health through various mechanisms, such as inflammation, oxidative stress, and immune system dysregulation [10, 11].

While the general population is at significant risk from air pollution, certain vulnerable populations, such as PLWH, may experience disproportionate health burdens due to the combined effects of environmental and biological factors [12, 13]. HIV infection and ART-related complications, such as immune suppression, chronic inflammation, and metabolic abnormalities, may increase susceptibility to the detrimental effects of air pollution [14, 15]. Despite this plausible connection, the specific interaction between air pollution exposure and mortality risk among PLWH remains an underexplored area in the literature. A better understanding of this association is essential, as it could inform public health policies and interventions aimed at reducing the burden of disease among this already vulnerable population.

This systematic review aims to address this gap in the literature by examining the association between exposure to air pollutants and the risk of mortality among PLWH. Specifically, it seeks to synthesize available evidence on the health effects of various air pollutants such as PM, NO_2, O_3, and CO on mortality risk in PLWH. By systematically evaluating the existing literature, this review will contribute to a more comprehensive understanding of the environmental factors that influence health outcomes among PLWH and provide insights into potential strategies for mitigating these risks.

Methods

The primary research question addressed by this systematic review was to explore the association between exposure to air pollutants and the risk of mortality among PLWH. Specifically, the review aimed to investigate the types of air pollutants, such as PM, NO_2, SO_2, O_3, and CO, that are associated with increased mortality risk among PLWH.

The primary research question addressed by this systematic review was to explore the association between exposure to air pollutants and the risk of mortality among PLWH. Specifically, the review aimed to investigate which types of air pollutants, such as PM, NO_2, SO_2, O_3, and CO, are associated with an increased mortality risk among PLWH. This systematic review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (Table S1) guidelines to ensure a comprehensive, transparent, and replicable process in assessing the association between air pollutant exposure and mortality risk among PLWH. The review was conducted in several stages: formulation of research questions, literature search, study selection, data extraction, and quality assessment. The protocol for this review has been registered in PROSPERO: CRD42024584352.

Literature search

A comprehensive and systematic search of the literature was conducted across multiple electronic databases, including PubMed, Web of Science, and Embase. The search included articles published up to August 2024. The search strategy incorporated a combination of Medical Subject Headings (MeSH) terms and free-text keywords to ensure a broad retrieval of relevant studies. The search terms included combinations of the following: "HIV," "people living with HIV," "AIDS," "air pollution," "particulate matter," "NO_2," "SO_2," "O_3," "CO," "mortality,"

"death," "comorbidities," and "cardiovascular risk." A full list of search terms and Boolean operators used for each database is provided in Table S2. No restrictions were placed on the geographical location or the demographic characteristics of study populations.

Eligibility criteria

The eligibility criteria for study selection were determined using the Population, Exposure, Comparison, and Outcome (PECO) framework. The population included PLWH of any age, gender, or ethnicity. The exposure criterion encompassed any form of air pollution such as PM, NO_2, SO_2, O_3, and CO. For comparison, studies that examined mortality rates among PLWH exposed to varying levels of air pollution or compared PLWH to a general or healthy population in relation to pollution exposure were included. The outcome focused on mortality, including all-cause mortality, HIV-related mortality, or mortality due to specific comorbidities, such as cardiovascular or respiratory diseases. Studies were included if they met the following criteria: [1] original research articles that quantitatively assessed the association between air pollution exposure and mortality in PLWH [2], cohort, case-control, or cross-sectional studies that measured air pollution exposure using tools like ambient air quality monitoring data and reported mortality outcomes, and [3] studies that provided sufficient data for calculating effect sizes, such as relative risk RR), odds ratio (OR), or hazard ratio (HR). The search was restricted to peer-reviewed articles published in English. Studies were excluded if they [1] focused exclusively on the general population or other chronic health conditions without separately reporting results for PLWH [2], did not report original data, such as narrative reviews, opinion pieces, or commentaries, or [3] were not published in peer-reviewed journals, such as conference abstracts.

Screening and study selection

All identified studies from the literature search were initially screened by reviewing their titles and abstracts to determine their relevance to the research question. Two independent reviewers performed the screening process to ensure objectivity and consistency. Studies that met the inclusion criteria, or those that required further evaluation based on their abstracts, were retrieved for full-text review. During the full-text screening, the same reviewers independently assessed each study against the predefined eligibility criteria, which were based on the PECO framework. Any discrepancies or disagreements between the reviewers were resolved through discussion. In cases where a consensus could not be reached, a third reviewer was consulted to provide a final decision. This multi-stage screening process ensured that only studies of high relevance and quality were included in the final analysis. A semi-automated software was used for screening the articles (Nested-Knowledge).

Data extraction

Data extraction was performed using a standardized form to ensure consistency and reduce bias, with two independent reviewers conducting the data extraction. Key information extracted from each study included study details such as author(s), year of publication, country, study design, and sample size, as well as air pollution exposure, including the types of pollutants measured, duration of exposure, and pollution levels. Mortality outcomes were also collected, focusing on mortality risk estimates such as RR, OR, or HR. The final dataset was then reviewed by a third reviewer to ensure completeness and accuracy.

Quality assessment

The quality and risk of bias of the included studies were assessed using the Newcastle-Ottawa Scale (NOS) for observational studies. The NOS evaluates studies on three main criteria: selection of the study groups, comparability of groups, and ascertainment of outcomes. Each study was awarded a score ranging from 0 to 9 based on these criteria. Studies scoring 7 or above were considered high quality, while those scoring between 5 and 6 were deemed moderate quality, and those scoring below 5 were considered low quality.

Data synthesis and analysis

Given the expected heterogeneity in the measurement of air pollution exposure, population characteristics, and mortality outcomes, a narrative synthesis of the findings was conducted. Quantitative data on mortality risk estimates were summarized using tables. Effect sizes for all-cause mortality and AIDS-related mortality were summarized in table form from multiple studies. The type of exposure, specific air pollutants, and relevant subgroups were also considered and reported in the tables.

Results

Literature search

The study selection process began with the identification of 464 records from various databases, including 108 from PubMed, 142 from Embase, and 214 from Web of Science. After removing 137 duplicate records, 327 records were screened for relevance. Following this screening process, 307 records were excluded. Subsequently, 20 reports were sought for full-text retrieval, all of which were successfully retrieved. Upon assessing these full-text reports, 11 articles were excluded 5 for being irrelevant and 6 for not reporting the outcome of interest. In the end, 9 studies [13, 16–23] were included in the systematic review (Fig. 1).

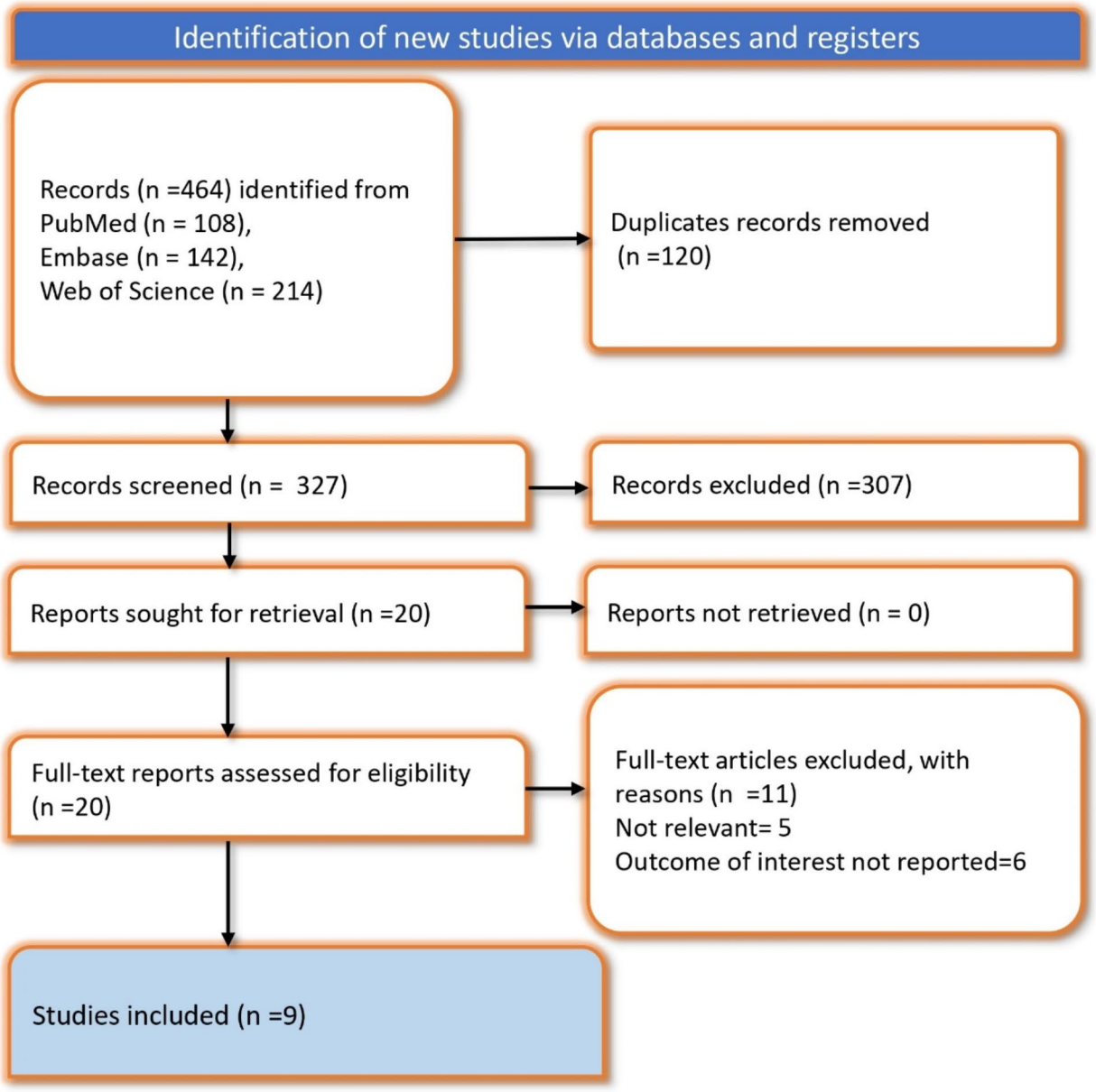

Fig. 1 PRISMA flowchart depicting article selection and screening process

Characteristics of included studies

The included studies predominantly focus on the impact of air pollution on HIV/AIDS-related mortality in China (Table 1). Of the nine studies reviewed, eight are longitudinal, case-crossover, or cohort studies, reflecting a strong preference for designs that can track changes over time and establish a more direct correlation between particulate exposure and health outcomes. The sample sizes vary widely, ranging from 1,467 to 170,655 participants, allowing for a broad examination of the effects across different demographic segments. The quality assessment of the studies is given in Table S3. Table 2 summarizes the results from included studies.

Association of air pollutants with AIDS-related mortality

The studies consistently show that exposure to PM is significantly associated with increased AIDS-related mortality among people living with HIV (PLHIV). In particular, Zhang et al. (2023-A) reported a 2.38% increase in HR for AIDS-related mortality per 1 μg/m^3 increment in PM1 concentration, with females (3.17%) and individuals aged 18–34 (2.36%) experiencing the highest risk.

Table 1 Characteristics of included studies and key findings

Author	Study design	Country	Sample size	Age (in years)	Male%	Follow up (in years)	Exposure measurement method	Key findings
Chen H 2024 (16)	Longitudinal study	China	7270	42.8	67.9	8	Daily maximum 8-hour average O_3 concentrations measured using data from the Tracking Air Pollution in China (TAP) platform, predicted with a three-stage random forest model	Long-term $PM_{2.5}$ exposure increases mortality risk among people living with HIV, especially at higher concentrations. PLWH with lower CD4 counts (< 200 cells/μl) are more vulnerable to air pollution's effects
Liang W 2024 (17)	Longitudinal study	China	7444	37.7	90.3	4.05	Average concentrations of PM_1, $PM_{2.5}$, PM_{10}, O_3, and NO_2 from the China High Air Pollutants dataset, estimated via space-time extremely randomized trees models	Long-term PM exposure is linked to increased AIDS-related deaths, with complications partially mediating this effect, especially in older PWHA
Sun D 2024(18)	Case-crossover study	China	1,467	NA	NA	NA	Daily mean SO_2, NO_2, and CO were generated by artificial intelligence algorithms combined with big data.	An increase in SO_2 was linked to higher AIDS-related death risk, especially in males and those over 65, with the warm season being more sensitive to SO_2 and NO_2 effects.
Yang S 2024 (13)	Prospective Cohort	China	170,655	44.6	70.6	NA	Combination of satellite-derived data, ground-level monitoring, and computational modelling	Ground-level O_3 exposure increases all-cause and AIDS-related mortality risk in PLWH, especially those with low CD4 + levels, due to their heightened susceptibility.
Zhang F-A 2023 (19)	Time-stratified case-crossover design	China	1,505 (deaths)	< 60 years	80	NA	Daily average concentrations of PM1, PM2.5, and PM10, obtained from the ChinaHighAirPollutants dataset with 1 km^2 resolution	PM1 increased the risk of ACD and ARD, with male AIDS patients being more vulnerable. PM_1 and $PM_{2.5}$ effects on ARD were stronger in patients over 60 and during colder months.
Zhang F-B 2023 (21)	Cohort study	China	28,140	> 18	78.1	3.8	Annual PM1 concentrations estimated using the Space-Time Extremely Randomized Trees model with data averaged by administrative region and extracted to 1 km^2 resolution.	Long-term exposure to PM1 was significantly associated with AIDS-related deaths and all-cause death among HIV/AIDS patients
Zhang F –C 2023 (22)	Cohort study	China	23,809	43.1	80.8	2.7	Annual concentrations of PM2.5 and PM10 from the ChinaHighAirPollutants dataset, which offers high-resolution (1 km × 1 km) ground-level air pollutant data across China	Increased risk of all-cause and AIDS-related deaths from long-term PM2.5 and PM10 exposure, with HIV-positive individuals over 60 being more vulnerable
Zhang F 2023 –D (20)	Case control study	China	1467	NA	79	NA	Annual concentrations of PM2.5 and PM10 from the ChinaHighAirPollutants dataset, which offers high-resolution (1 km × 1 km) ground-level air pollutant data across China	A 1 IQR increase in O3 concentration, lagged by 4 days, was associated with a 15% increase in AIDS-related deaths, with males showing higher susceptibility
Zhu S 2024 (23)	Cohort study	China	28,140	42.4	78.1	4.7	Annual concentrations of PM2.5 and its components (sulfate, nitrate, ammonium, organic matter, black carbon) from the TAP dataset, with exposure assigned based on participants' geocoded residences.	PM2.5, particularly NH4 + and $SO4^2$–, increases death risk in PLWHA, with those aged 65 + being especially vulnerable.

Abbreviations:- ACD (All-Cause Deaths), ARD (AIDS-Related Deaths), CD4 (Cluster of Differentiation 4), IQR (Interquartile Range), NA (Not Available), NH4+ (Ammonium), NO2 (Nitrogen Dioxide), O3 (Ozone), PLWH (People Living with HIV), PLWHA (People Living with HIV/AIDS), PM (Particulate Matter), PM1 (Particulate Matter with a diameter≤1 μm), PM2.5 (Particulate Matter with a diameter≤2.5 μm), PM10 (Particulate Matter with a diameter≤10 μm), PWHA (People With HIV/AIDS), SO2 (Sulfur Dioxide), $SO4^2$– (Sulfate)

Males also showed a notable increase (2.22%), but their risk was slightly lower compared to females. The study also highlighted that individuals with a lower CD4 count (<500 cells/μL) were at a higher risk (2.51%) compared to those with a higher CD4 count (>500 cells/μL), who did not show a significant association. This suggests that immunocompromised PLHIV may be more vulnerable to the adverse effects of air pollution. Similarly, Zhang et al. (2023-B) found that exposure to $PM_{2.5}$ was associated with a 1.65% increase in HR for AIDS-related mortality. Subgroup analysis revealed that older individuals (>60 years) were particularly vulnerable, with a 2.66% increase in HR, whereas younger individuals (<60 years) exhibited a lower but still significant risk (1.17%). The effects of PM_{10} were comparatively weaker, with only a 0.90% increase in HR, and the impact was more pronounced in females (1.21%) and older individuals (1.62%). Liang et al. (2024) provided additional evidence that long-term exposure to PM_1 significantly increases the risk of AIDS-related mortality, reporting a 2.1% increase in HR for every 1 μg/m³ increment. The study also found that older individuals (>60 years) were more susceptible, with a 5.2% increase in HR, emphasizing the compounding effects of age and long-term exposure.

Short-term exposure studies further corroborated these findings. Zhang et al. (2023-D) demonstrated that short-term exposure to PM_1 at lag 0–4 days increased the OR for AIDS-related mortality by 3.13% in males and 4.08% in individuals over 60. This acute exposure effect highlights the immediate risk posed by elevated PM_1 levels, particularly for older populations. Interestingly, females and younger populations (<60 years) did not show significant short-term effects, suggesting potential differences in susceptibility based on age and gender. Additionally, females appeared to be more affected by PM1 exposure than males, with a 4.1% versus 2.0% increase in HR, respectively, suggesting potential biological differences in response to particulate matter exposure.

The impact of O3 on AIDS-related mortality was also highlighted in the studies. Zhang et al. (2023-C) reported that a 1.36% increase in OR was observed per interquartile range (IQR) increase in O3 concentration in individuals over 65 years, while younger individuals showed a more modest risk increase (1.10%). Seasonal variation was also noted, with a higher risk observed during the warm season (1.29%) compared to the cold season (0.97%), which may be due to increased ozone concentrations during warmer months.

Sun et al. (2024) explored the effects of gaseous pollutants, finding that SO_2 was associated with a 1.17% increase in OR for AIDS-related mortality, though no significant effect was observed for all-cause mortality.

Impact of air pollutants on all-cause mortality

In addition to AIDS-related mortality, the studies also explored the relationship between air pollutant exposure and all-cause mortality among PLHIV. Zhang et al. (2023-A) reported that while PM_1 exposure was strongly associated with AIDS-related deaths, its association with all-cause mortality was more modest, with a 0.69% increase in HR per 1 μg/m³ increase in PM_1 concentration. Subgroup analyses revealed that males (0.84%) were slightly more affected than females (0.27%), and younger individuals (0.70%) had a higher risk than older individuals (1.15%).

Exposure to $PM_{2.5}$ was similarly linked to a 0.69% increase in HR for all-cause mortality, with males (0.66%) and older individuals (0.62%) showing higher susceptibility. Zhang et al. (2023-B) found that PM_{10} had a smaller effect on all-cause mortality, with a 0.39% increase in HR across the total population. However, females (0.48%) and those under 60 (0.31%) demonstrated slightly higher risk compared to their counterparts. The modest association of PM_{10} with all-cause mortality could reflect its larger particle size, which may not penetrate as deeply into the respiratory system as $PM_{2.5}$ and PM_1, resulting in less severe health outcomes.

In short-term exposure studies, PM_1 and $PM_{2.5}$ showed limited effects on all-cause mortality. Zhang et al. (2023-D) found that short-term exposure to PM1 increased all-cause mortality by 1.11% in males, while females and younger populations did not exhibit significant increases. Similarly, short-term $PM2._5$ exposure resulted in a 0.53% increase in HR for all-cause mortality in males, but the effects were weaker in other subgroups.

Conversely, ozone exposure had a more consistent impact on all-cause mortality. Chen et al. (2024) demonstrated that a 10 μg/m³ increase in O_3 concentration was associated with a 1.11–1.13% increase in HR for all-cause mortality across different lag periods (1–7 days).

Chemical constituents of particulate matter and specific pollutants

Beyond the general association with particulate matter, several studies focused on the chemical constituents of PM and their specific impacts on mortality. Zhu et al. (2024) investigated the long-term effects of fine particulate matter components, such as sulfate ($SO4^{2-}$), nitrate ($NO3^-$), and ammonium ($NH4^+$). The study found that $NH4^+$ was associated with the highest increase in HR for both AIDS-related (5.13%) and all-cause mortality (2.97%). $NO3^-$ and $SO4^{2-}$ also contributed to elevated risks, with a 2.22% and 2.23% increase in HR for AIDS-related mortality, respectively.

Interestingly, exposure to black carbon and organic matter did not show significant associations with mortality. Black carbon was associated with a 4.62% increase in

Table 2 Summary of results of studies investigating the association between air pollution exposure and mortality in PLWH

Study	Air pollutant	Exposure definition	Subgroups	Type of effect estimate	AIDS related mortality	All-cause mortality
Zhang 2023 -A (21)	PM1	1 µg/m3 increase of the concentration of PM	Total	% change in HR (95% CI)	2.38 (1.62 to 3.15)	0.69 (0.22 to1.17)
			Male		2.22 (1.35 to 3.10)	0.84 (0.31 to1.38)
			Female		3.174 (1.55 to4.81)	0.27 (-0.76 to 1.31)
			18–34 years old		2.36 (1.41 to 3.31)	0.70 (0.09 to 1.30)
			35–64 years old		1.70 (-0.06 to3.48)	1.15 (-0.01 to 2.33)
			CD4 < 500 cells/µL		2.51 (1.72 to 3.31)	0.97 (0.46 to 1.47)
			CD4 > 500 cells/µL		2.02 (-1.30 to 5.45)	-0.42 (-1.81 to 0.98)
Zhang 2023 –B (22)	PM2.5	1 µg/m3 increase of the concentration of PM	Total	% change in HR (95% CI)	1.65 (1.14 to 2.17)	0.69 (0.39 to 1.00)
			Male		1.56 (0.99 to 2.13)	0.66 (0.32 to 0.99)
			Female		2.09 (0.90 to 3.30)	0.85 (0.13 to 1.58)
			< 60 years old		1.17 (0.56 to 1.80)	0.55 (0.17 to 0.93)
			> 60 years old		2.66 (1.76 to 3.58)	0.62 (0.12 to 1.13)
	PM10	Increase of 1 µg/m3 in PM10 concentration	Total		0.90 (0.56 to 1.24)	0.39 (0.18 to 0.59)
			Male		0.82 (0.44 to 1.20)	0.37 (0.14 to 0.59)
			Female		1.21 (0.44 to 1.99)	0.48 (0.01 to 0.96)
			< 60 years old		0.57 (0.17 to0.98)	0.31 (0.06 to 0.56)
			> 60 years old		1.62 (1.01 to2.23)	0.34 (0.00 to 0.69)
Zhang 2023 –C (20)	O3	Per IQR increased in O3 at lag 4 days	Male	% change in OR (95% CI)	1.20 (1.04 to1.37)	NA
			Female		0.98 (0.73 to 1.32)	NA
			< 65years old		1.10 (0.96 to 1.27	NA
			> 65 years old		1.36 (1.05 to 1.76)	NA
			Warm season		1.29 (1.09 to 1.52)	NA
			Cold season		0.97 (0.80 to 1.18)	NA
Zhang 2023 –D (19)	PM1	Per 1 µg/m3 increased in ambient PM1 at lag 0–4 days	Male	% change in OR (95% CI)	3.13 (1.33 to 4.96)	1.11 (0.14 to 2.10)
			Female		-3.51 (-8.95 to 2.26)	-0.36 (-2.56 to 1.89)
			< 60 years old		1.42 (-0.78 to 3.67)	0.74 (-0.45 to 1.94)
			> 60 years old		4.08 (1. 27 to 6.96)	0.53 (-0.16 to 1.24)
			Warm season		4.49 (-0.35 to 9.56)	-0.34 (-1.62 to 0.97)
			Cold season		2.30 (0.50 to 4.13)	0.57 (0.06 to 1.07)
	PM2.5	Per 1 µg/m3 increased in ambient PM2.5 at lag 0–4 days	Male	% change in OR (95% CI)	1.59 (0.61 to2.57)	0.53 (0.01 to 1. 05)
			Female		-1.33 (-4.06 to 1.49)	-0.10 (-1.21 to 1.02)
			< 60 years old		0.63 (-0.56 to 1.85)	0.31 (-1.62 to 0.97)
			> 60 years old		2.06 (0.59 to 3.55)	0.53 (-0.16 to 1.24)
			Warm season		1.61 (-0.94 to 4.22)	-0.34 (-1.62 to 0.97)
			Cold season		1.19 (0.21 to 2.17)	0.57 (0.06 to 1.07)
	PM10	per 1 µg/m3 increased in ambient PM10 at lag 0–4 days	Male	% change in OR (95% CI)	0.95 (0.27 to 1.63)	0.21 (-0.15 to 0.57)
			Female		-2.07 (-4.26 to 0.17)	-0.54 (1.40 to 0.33)
			< 60 years old		0.33 (-0.47 to 1.14)	0.12 (-0.30 to 0.55)
			> 60 years old		1.21 (0.11 to 2.32)	0.04 (-0.47 to 0.57)
			Warm season		0.58 (-0.70 to 1.87)	-0.23 (-0.84 to 0.39)
			Cold season		0.67 (-0.07 to 1.42)	0.27 (-0.12 to 0.66)

Table 2 (continued)

Study	Air pollutant	Exposure definition	Subgroups	Type of effect estimate	AIDS related mortality	All-cause mortality
Liang 2024 (17)	PM1	Per 1 μg/m3 increment of PM1 in long term	Total	HR (95% CI)	1.021 (1.009 to 1.033)	NA
			Male		1.020 (1.008 to 1.032)	NA
			Female		1.041 (1.011 to 1.072)	NA
			< 60 years old		1.024 (1.011 to 1.037)	NA
			> 60 years old		1.052 (1.032 to 1.073)	NA
			BMI < 24		1.023 (1.011 to 1.035)	NA
			BMI ≥ 24		1.055 (1.017 to 1095)	NA
	PM2.5	Per 1 μg/m3 increment of PM2.5 in long term	Total	HR (95% CI)	1.012 (1.005 to 1.020)	NA
			Male		1.012 (1.004 to 1.019)	NA
			Female		1.027 (1.008 to 1.046)	NA
			< 60 years old		1.015 (1.007 to 1.023)	NA
			> 60 years old		1.031 (1.019 to 1.043)	NA
			BMI < 24		1.014 (1.006 to 1.021)	NA
			BMI ≥ 24		1.036 (1.012 to 1.060)	NA
	PM10	Per 1 μg/m3 increment of PM10 in long term	Total	HR (95% CI)	1.010 (1.005 to 1.015)	NA
			Male		1.010 (1.005 to 1.015)	NA
			Female		1.017 (1.004 to 1.030)	NA
			< 60 years old		1.011 (1.006 to 1.017)	NA
			> 60 years old		1.023 (1.014 to 1.032)	NA
			BMI < 24		1.011 (1.008 to 1.014)	NA
			BMI ≥ 24		1.027 (1.021 to 1.032)	NA
Zhu 2024 (23)	PM2.5	Long-term exposure to PM2.5	Total	% change in HR (95% CI)	0.32 (0.01 to 0.64)	0.02 (− 0.19 to 0.22)
	SO4 2-	Long-term exposure to SO4 2-	Total		2.23 (0.78 to 3.69)	0.49 (− 0.45 to 1.44)
	NO3 −	Long-term exposure to NO3 −	Total		2.22 (0.67 to 3.80)	1.56 (0.55 to 2.58)
	NH4 +	Long-term exposure to NH4 +	Total		5.13 (2.89 to 7.43)	2.97 (1.52 to 4.44)
	Black carbon	Long-term exposure to BC	Total		4.62 (− 0.69 to 10.21)	2.56 (− 5.98 to 0.98)
	Organic matter	Long-term exposure to OM	Total		0.69 (− 0.61 to 2.00)	0.47 (− 1.32 to 0.39)
Chen 2024 (16)	O3	10 μg/m3 increase in ambient O3 concentration	Lag 1 days	OR (95% CI)	NA	1.11 (1.04 to 1.18)
		10 μg/m3 increase in ambient O3 concentration	Lag 2 days		1.08 (1.00 to 1.17)	1.08 (1.01 to 1.15)
		10 μg/m3 increase in ambient O3 concentration	Lag 3 days		NA	1.08 (1.02 to 1.15)
		10 μg/m3 increase in ambient O3 concentration	Lag 4 days		1.08 (1.01 to 1.17)	1.07 (1.00 to1.13)
		10 μg/m3 increase in ambient O3 concentration	Lag 5 days		1.12 (1.05 to 1.21)	1.07 (1.01 to 1.14)
		10 μg/m3 increase in ambient O3 concentration	Lag 6		1.09 (1.01 to 1.17)	NA
		10 μg/m3 increase in ambient O3 concentration	7-day moving average		1.15 (1.04 to 1.27)	1.13 (1.04 to 1.22)

Table 2 (continued)

Study	Air pollutant	Exposure definition	Subgroups	Type of effect estimate	AIDS related mortality	All-cause mortality
Yang 2024 (13)	PM2.5	PM2.5 [10–35 μg/m^3]	3-years RD	RD% (95% CI)	NA	−3.23 (−3.56 to −2.93)
		PM2.5 [35–50 μg/m^3]			NA	−0.05 (−0.32 to 0.20)
		PM2.5 [50–75 μg/m^3]			NA	3.59 (3.19 to 4.05)
		PM2.5 [10–35 μg/m^3]	5-years RD		NA	−4.06 (−4.47 to −3.62)
			5-years RD			
		PM2.5 [35–50 μg/m^3]	5-years RD		NA	0.44 (0.07 to 0.800
		PM2.5 [50–75 μg/m^3]			NA	5.04 (4.42 to 5.61)
Sun 2024 (18)	SO2	Per interquartile range increase in concentration SO2 at lag 4 day	Total	OR (95% CI)	1.17 (1.01 to 1.35)	NA

Abbreviations:- BC (Black Carbon), BMI (Body Mass Index), CD4 (Cluster of Differentiation 4), CI (Confidence Interval), HR (Hazard Ratio), IQR (Interquartile Range), NA (Not Available), NH4+ (Ammonium), NO3− (Nitrate), O3 (Ozone), OM (Organic Matter), OR (Odds Ratio), PM (Particulate Matter), PM1 (Particulate Matter with a diameter ≤ 1 μm), PM2.5 (Particulate Matter with a diameter ≤ 2.5 μm), PM10 (Particulate Matter with a diameter ≤ 10 μm), RD (Risk Difference), SO2 (Sulfur Dioxide), $SO4^{2}$− (Sulfate)

AIDS-related mortality, but the confidence intervals were wide, indicating uncertainty in the results. Organic matter exposure had a weak and non-significant association with both AIDS-related and all-cause mortality.

Mortality risk based on exposure levels and hypothetical intervention scenarios

Yang et al. (2024) provided a unique perspective by assessing the impact of different levels of $PM_{2.5}$ exposure on all-cause mortality risk under hypothetical intervention scenarios. The study found that low to moderate levels of $PM_{2.5}$ exposure (10–35 μg/m^3) were associated with a reduction in mortality risk over 3 and 5 years, with risk differences of −3.23% and −4.06%, respectively. However, at higher exposure levels (50–75 μg/m^3), the mortality risk significantly increased, with a 3.59–5.04% rise.

Discussion

This systematic review aimed to assess the association between exposure to air pollutants and mortality risk among PLWH. The impact of these environmental stressors appears to be influenced by several demographic and clinical factors, including age, gender, immune status, and seasonality.

The studies reviewed consistently demonstrate that particulate matter, particularly fine PM (PM_1 and $PM_{2.5}$), is significantly associated with both AIDS-related and all-cause mortality among PLWH. Zhang et al. [19–21] reported that even modest increases in PM_1 and $PM_{2.5}$ concentrations were associated with elevated HRs for mortality. The association was most pronounced in subgroups such as females, older adults, and individuals with lower CD4 counts. Liang et al. [17] further reinforced these findings, showing a cumulative impact of long-term exposure to PM1, which exacerbates mortality risk, particularly in older individuals and those with a higher body mass index (BMI).

The more profound effect of PM1 and $PM_{2.5}$ compared to PM_{10} is noteworthy. This can be attributed to the smaller size of PM1 and $PM_{2.5}$ particles, which allows them to penetrate deeper into the respiratory tract, reaching the alveoli and potentially entering the bloodstream [24, 25]. These smaller particles can cause systemic inflammation and oxidative stress, exacerbating underlying comorbidities in PLWH [26].

Age also emerged as a significant factor, with younger individuals (<60 years) showing a relatively lower but still notable risk of mortality due to PM exposure compared to older individuals. The stronger association observed in older PLWH, particularly those over 60 years of age, aligns with the established literature on air pollution and its disproportionate impact on vulnerable populations [27, 28]. Aging is associated with a natural decline in immune function and an increased prevalence of chronic conditions, making older individuals more susceptible to the harmful effects of air pollution [29]. Furthermore, PLWH are known to experience premature aging and immune senescence, even when treated with ART, which may further amplify their vulnerability to air pollution [30, 31]. Younger PLWH may have a more resilient immune system and fewer underlying comorbidities than their older counterparts, explaining their lower risk. However, the fact that younger individuals were still significantly affected suggests that HIV infection itself, along with ART and chronic inflammation, may exacerbate the health effects of air pollution even in younger populations [32].

Gender disparities in the effects of air pollution were consistently observed, with females showing higher susceptibility to PM_1 and $PM2._5$ exposure in several studies [17, 22]. This may be partially explained by differences

in lung function, hormonal responses, and occupational exposure patterns between males and females. Research suggests that females tend to have a smaller airway diameter and reduced lung capacity, which may increase the deposition of fine particles and heighten their susceptibility to respiratory conditions [33]. Moreover, hormonal factors, such as estrogen, are known to modulate immune responses and may interact with the inflammatory processes induced by particulate matter.

Seasonal variations in the impact of air pollutants on mortality were highlighted in several studies. Zhang et al. [19, 20] and Sun et al. [18] found that mortality risks associated with both particulate matter and ozone were higher during the warm season. This may be due to increased pollutant concentrations during warmer months [34, 35], driven by factors such as higher temperatures and photochemical reactions that produce ozone and other secondary pollutants. In colder seasons, the risk was lower but still significant, indicating that PLWH are at risk year-round, though the degree of exposure and impact may vary with seasonality [32].

Short-term exposure studies also revealed interesting findings. While long-term exposure to air pollution was consistently associated with elevated mortality risks, short-term peaks in air pollution, particularly PM1 and ozone, were also linked to increased deaths. Zhang et al. [20] showed that even brief increases in PM1 exposure, lasting just a few days, could lead to a significant rise in AIDS-related and all-cause mortality. This finding is critical, as it underscores the importance of mitigating not only chronic pollution but also episodic spikes in pollutant levels, which can have immediate and severe health consequences for PLWH.

Several studies [18, 23] investigated the chemical constituents of particulate matter, such as $NH4^{+}$, $SO4^{2-}$, $NO3^{-}$, and black carbon, and their specific impact on mortality in PLWH. The results suggest that $NH4^{+}$ and $SO4^{2-}$ were the most harmful components, contributing significantly to both AIDS-related and all-cause mortality. These constituents are typically generated by industrial activities, vehicle emissions, and agricultural processes, indicating that certain sources of pollution may be particularly detrimental to PLWH. Black carbon, a marker of traffic-related air pollution, was also associated with increased mortality risk. Organic matter, another component of particulate matter, did not show a significant association with mortality, suggesting that not all components of PM are equally harmful. The findings on the chemical constituents of particulate matter are important because they highlight the need for targeted air quality interventions that address the most harmful pollutants. Policies aimed at reducing emissions from industrial activities, transportation, and agriculture could have a significant impact on reducing mortality risks in PLWH, especially in regions where these pollutants are prevalent.

Particles such as O_3 and SO_2 also emerged as significant pollutants in relation to mortality risk in PLWH. Zhang et al. [22] and Chen et al. [16] found that short-term increases in ozone concentrations were associated with elevated ORs for mortality, particularly in males and older individuals. Ozone is a powerful oxidant that can cause airway inflammation, reduce lung function, and exacerbate chronic respiratory conditions. The fact that PLWH are particularly susceptible to ozone's effects may be due to their compromised immune function and pre-existing respiratory issues, which are common in this population. SO_2, a byproduct of fossil fuel combustion, was also linked to increased mortality risk, particularly for AIDS-related deaths [18]. SO_2 is known to irritate the respiratory system and cause bronchoconstriction, especially in individuals with pre-existing lung conditions. For PLWH, who may already have compromised lung function due to opportunistic infections or ART-related side effects, SO_2 exposure may exacerbate respiratory issues and increase mortality risk.

The findings from this review highlight critical public health policy implications for reducing mortality among PLWH in relation to air pollution exposure. Strengthening air quality standards to limit pollutants such as PM_1, $PM_{2.5}$, ozone, and sulfur dioxide is essential, particularly in urban centers with high concentrations of PLWH, where pollution levels tend to be higher [36]. Regulatory efforts should target emissions from industrial sources, transportation, and agriculture, which are primary contributors to these pollutants. Additionally, targeted interventions are crucial for protecting vulnerable subgroups, including older adults, females, and individuals with lower CD4 counts, such as providing air quality alerts, promoting the use of respiratory protection on high-pollution days, and encouraging the use of indoor air filtration systems in affected areas. Healthcare providers should integrate awareness of air pollution risks into their care for PLWH at higher risk e.g. older age, smokers, those with chronic pulmonary comorbidities, etc. Advising these vulnerable patients on strategies to minimize exposure and closely monitoring for respiratory and cardiovascular symptoms that may be exacerbated by poor air quality in these vulnerable PLWH population. Proactive interventions, early symptom management, and counseling can significantly mitigate the health risks associated with air pollution [37–40]. It's essential to concurrently tackle other prevalent risk factors that compound the effects of environmental pollutants on lung health and non-communicable diseases (NCDs). Smoking, a widespread behavior among PLWH, markedly exacerbates their vulnerability to respiratory conditions, cardiovascular diseases, and various forms of cancer

conditions to which PLWH are already predisposed due to chronic immune system challenges and ART-related complications [41, 42]. This synergy between smoking and air pollution can lead to a compounded deterioration in lung function and an accelerated progression of comorbidities. Therefore, it is imperative that public health strategies not only focus on reducing exposure to air pollutants but also aggressively address tobacco use within this population. Integrative interventions should include cessation programs tailored to the specific needs of PLWH, considering the psychological and social dynamics that may affect their smoking habits [43, 44]. Policymakers should enforce stricter emissions regulations and promote renewable energy. Enhancing air quality monitoring and alert systems, improving indoor ventilation, and creating more green spaces are key measures. Public health campaigns can also raise awareness about pollution risks and protective strategies, making these findings more relevant to public health practice.

The association between air pollution and mortality among PLWH is underpinned by complex biological mechanisms that involve immune suppression and the exacerbation of inflammatory responses. Air pollutants, particularly PM, NO_2, and ozone, can significantly impair immune function, making PLWH more susceptible to infections and other health complications [45]. Exposure to these pollutants has been shown to enhance pro-inflammatory cytokine production, such as interleukin-6 (IL-6), which can lead to chronic inflammation and immune dysregulation. For instance, air pollution can stimulate T helper lymphocyte type 2 (Th2) and Th17 responses, which are associated with allergic reactions and chronic inflammatory diseases, further complicating the health landscape for PLWH [46, 47]. Moreover, pollutants can induce oxidative stress, a condition characterized by an imbalance between reactive oxygen species (ROS) production and antioxidant defences [48]. This oxidative stress not only triggers inflammatory pathways but also disrupts normal immune responses, leading to increased vulnerability to respiratory infections. The inhalation of air pollutants can damage respiratory epithelial cells, compromising the mucosal barrier and facilitating pathogen entry. Studies have indicated that long-term exposure to air pollutants correlates with reduced levels of protective proteins like alpha-1-antitrypsin, which plays a crucial role in modulating inflammatory responses. Consequently, this combination of immune suppression and heightened inflammatory responses may contribute to increased morbidity and mortality rates among PLWH exposed to elevated levels of air pollution [49]. Understanding these pathways is essential for developing targeted interventions aimed at mitigating the health impacts of air pollution on this vulnerable population.

The implications of our findings are significant for public health policy, especially in regions with high HIV prevalence and poor air quality. The established link between air pollution exposure and increased mortality risk among PLWH shows the urgent need for integrated health and environmental policies. Health systems should consider air quality as a key component of managing the health of PLWH, incorporating regular monitoring of air pollution levels into their care strategies. Additionally, interventions such as improving air filtration in homes, promoting access to cleaner transportation options, and increasing green spaces in urban areas could substantially mitigate the adverse health impacts of pollution on this vulnerable population. Public health campaigns should also aim to raise awareness among PLWH about the risks of air pollution and provide guidance on protective behaviors, especially during periods of high pollution levels. Legislative efforts aimed at reducing emissions and improving overall air quality are essential, ensuring that environmental health is part of comprehensive care and prevention strategies for PLWH. For future research, it is crucial to delve deeper into the specific biological mechanisms through which air pollution impacts PLWH, particularly how different pollutants influence immune function and inflammation pathways. Longitudinal studies and experimental designs would be beneficial to establish causal relationships and observe the long-term health outcomes of pollution exposure in this vulnerable population. There is a need to broaden the scope of studies to encompass diverse geographic and socioeconomic contexts, particularly in regions where both HIV prevalence and air pollution are significant public health concerns. This expanded focus will help to develop targeted interventions that are culturally and regionally adapted. Investigating the effectiveness of various air quality improvement interventions, such as the use of air filtration systems and changes in urban infrastructure, could provide actionable data to guide public health policies and protective measures for PLWH.

This systematic review encounters several limitations that could influence the interpretation of the findings. Primarily, the inclusion of studies predominantly conducted in China restricts the applicability of the results to other regions, particularly low- and middle-income countries with differing environmental conditions and healthcare systems. The heterogeneity in study designs, methods of exposure assessment, and the diversity of outcome measures complicates the synthesis of data and hampers the ability to conduct a robust meta-analysis or draw uniform conclusions across studies. Additionally, the reliance on ambient air quality data for exposure assessment could lead to misclassification of individual exposure levels, as these measurements may not accurately reflect personal exposure. This limitation is further

compounded by the potential variability in air pollution composition and concentration within different areas of the same city or region. Moreover, while our review identifies significant associations between air pollution and increased mortality risk in PLWH, it cannot establish causality. Many of the included studies did not adequately control for confounding factors such as socioeconomic status, access to healthcare, and adherence to ART, which can significantly influence health outcomes. These confounders are crucial, especially in studies involving vulnerable populations such as PLWH, who may face systematic disparities in health access and outcomes.

To enhance the robustness of future research, studies need to be conducted in diverse geographic settings, including countries with varying levels of pollution and healthcare infrastructure. It is also essential to employ standardized methods for measuring both air pollution exposure and health outcomes to facilitate more accurate comparisons and meta-analyses. Addressing these gaps will provide a more comprehensive understanding of how air pollution impacts mortality among PLWH globally and inform targeted interventions that can mitigate these effects in specific populations.

Conclusion

A significant association exists between exposure to air pollutants, particularly fine particulate matter and ozone, and increased mortality risk among PLWH. The findings emphasize the need for targeted public health interventions to reduce air pollution exposure, particularly in vulnerable subgroups such as older adults, females, and individuals with compromised immune function. Reducing air pollution levels and integrating environmental health considerations into HIV care could substantially improve health outcomes for this population, which continues to face disproportionate health burdens. Future research should focus on elucidating the mechanisms of air pollution's impact on health in PLWH and developing effective strategies to mitigate these risks.

Supplementary Information

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Supplementary Material 1

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Author contributions
Muhammed Shabil and Mahalaqua Nazli Khatib contributed to the conceptualization and design of the study. Bijaya Kumar Padhi, Prakasini Satapathy, Sakshi Pandey, Ashok Kumar Balaraman, Manvinder Brar, and Ganesh Bushi conducted data collection and analysis. Diptismita Jena and Mahendra Pratap Singh performed statistical analysis and data interpretation. Suhas Ballal, Pooja Bansal, Nagavalli Chilakam, Rachana Mehta and Kiran Bhopte assisted with manuscript drafting and revisions. Abhay M Gaidhane and Afukonyo Shidoiku Daniel provided supervision and critical revisions. Balvir S. Tomar, Ayash Ashraf, M Ravi Kumar, Ashish Singh Chauhan, and Sanjit Sah reviewed the manuscript for important intellectual content. All authors read and approved the final manuscript.

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The data is with the authors and available on request.

Declarations

Human ethics and Consent to participate declarations
Not applicable

Consent to Participate
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The authors declare no competing interests.

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