

REVIEW



## Air pollution and cardio-oncology: A converging health crisis

Pratyush Malik

Department of Biotechnology, KIIT School of Biotechnology, KIIT University, Bhubaneswar, Odisha

### ABSTRACT

Air pollution causes millions of deaths every year, making it a serious global health concern. Its effects go beyond respiratory problems; it has a significant impact on cancer outcomes and cardiovascular health. Cancer patients, who are especially susceptible to the cardiovascular effects of contaminated air, face additional risks, according to recent studies. The incidence and mortality of cardiovascular diseases among cancer patients have been linked to exposure to fine particulate matter (PM<sub>2.5</sub>), which exacerbates health disparities. With a focus on the ways that pollutants worsen cardiovascular conditions in cancer patients, this review explores the complex relationship between air pollution, cardiovascular diseases, and oncology. In order to reduce these compounded risks, it also promotes preventive measures and talks about the implications for public health.

### KEYWORDS

Heart disease;  
Cardio-oncology; Air  
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## Introduction

With major effects on oncology and cardiovascular health, air pollution has become a major global health concern. The widespread effects of air pollution on human health are highlighted by the World Health Organization's estimate that it causes about 7 million preventable deaths each year. Although air pollution's harmful effects on respiratory illnesses are widely known, its ability to exacerbate cardiovascular conditions and affect the course of cancer has recently come to light [1].

Particulate matter (PM), nitrogen oxides (NO<sub>x</sub>), sulfur dioxide (SO<sub>2</sub>), ozone (O<sub>3</sub>), and other harmful substances make up the complex mixture that is ambient air pollution. The capacity of fine particulate matter (PM<sub>2.5</sub>) to enter the systemic circulation and penetrate deeply into the lungs, thereby impacting multiple organ systems, makes it particularly concerning among these. Long-term exposure to PM<sub>2.5</sub> has been strongly linked in epidemiological studies to an increased risk of cardiovascular conditions, such as hypertension, atherosclerosis, myocardial infarction, and stroke. The immediate risks posed by environmental pollutants are further highlighted by recent research showing that even brief exposure to high levels of air pollution can adversely affect cardiovascular health. Air pollution has been linked in oncology to the etiology and development of several malignancies [2]. Interestingly, research has shown a link between higher levels of PM<sub>2.5</sub> and a higher risk of lung cancer in nonsmokers, indicating that environmental variables are important in the development of cancer. Furthermore, new data suggests a connection between air pollution and other cancers, such as breast cancer, where greater incidence rates have been linked to increased exposure to PM<sub>2</sub> [3].

One group that is especially at risk from the negative impacts of air pollution is cancer survivors. Although survival rates have increased due to advancements in cancer medicines, many survivors still suffer from chronic illnesses, particularly cardiovascular disorders, as a result of cardiotoxic cancer

treatments like radiation therapy and chemotherapy. Air pollution exposure may increase the risk of adverse cardiovascular events in this group by exacerbating pre-existing cardiovascular susceptibility. Furthermore, pulmonary toxicity brought on by several cancer therapies may increase vulnerability to respiratory problems linked to pollution [4].

Developing comprehensive public health interventions to mitigate these compounded risks requires an understanding of the interaction of cardiovascular health, cancer, and air pollution. The purpose of this review is to clarify the complex relationship between air pollution and cancer and cardiovascular disorders, with an emphasis on the increased susceptibility of cancer survivors. We want to provide guidance for focused initiatives and legislative actions that tackle this important environmental health issue by combining the available data [5,6].

### Cardiovascular Effects of Air Pollution

There is strong evidence that air pollution has a negative impact on cardiovascular health, making it a serious environmental health risk. Numerous pollutants, including as carbon monoxide (CO), sulfur dioxide (SO<sub>2</sub>), nitrogen dioxide (NO<sub>2</sub>), particulate matter (PM), and ozone (O<sub>3</sub>), have been linked to the onset and aggravation of cardiovascular diseases (CVDs). The effects of these pollutants on heart function, both directly and indirectly, are examined in this section.

### Particulate matter

Based on aerodynamic dimension, particulate matter is divided into three categories: coarse particles (PM<sub>10</sub>), fine particles (PM<sub>2.5</sub>), and ultrafine particles. Because it may enter the bloodstream and pierce deeply into the respiratory system, PM<sub>2.5</sub>, which have dimensions smaller than 2.5 micrometers, provides the greatest risk. Atherosclerosis, cardiac failure, arrhythmias, and hypertension have all been linked to exposure to PM<sub>2.5</sub>. Systemic inflammation, oxidative stress, endothelial

\*Correspondence: Mr. Pratyush Malik, Department of Biotechnology, KIIT School of Biotechnology, KIIT University, Bhubaneswar, e-mail: [2261091@biotech.kiit.ac.in](mailto:2261091@biotech.kiit.ac.in)

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dysfunction, and autonomic nervous system instability are some of the causes. For example, exposure to PM<sub>2.5</sub> can raise blood pressure and encourage the development of atherosclerotic plaque, which increases the risk of myocardial infarction and stroke [7,8].

### Nitrogen dioxide

One major source of air pollution in cities is NO<sub>2</sub>, which is mostly generated by industrial activities and automobile emissions. Increased hospitalizations for ischemic heart disease and heart failure have been associated with brief exposure to NO<sub>2</sub>. It is believed that inflammation and oxidative stress cause endothelial dysfunction and arterial stiffness, which have detrimental cardiovascular consequences. Furthermore, exposure to NO<sub>2</sub> may make pre-existing cardiovascular disorders worse by making the myocardium more vulnerable to ischemia damage [9].

### Sulfur dioxide

The burning of fossil fuels and industrial processes release SO<sub>2</sub>. Vasoconstriction and bronchoconstriction brought on by acute SO<sub>2</sub> exposure can have an indirect impact on heart function by raising cardiac workload. Long-term exposure has been linked to higher blood pressure and a higher risk of cardiovascular events. The fundamental processes that lead to endothelial dysfunction and atherosclerosis include oxidative stress and systemic inflammation [10].

**Table 1.** Common air pollutants and their cardiovascular effects.

Pollutant	Major Sources	Cardiovascular Impact	Associated Diseases
PM <sub>2.5</sub>	Vehicle emissions, industry	Inflammation, oxidative stress	Atherosclerosis, MI
NO <sub>2</sub>	Fossil fuel combustion	Endothelial dysfunction	Hypertension, stroke
O <sub>3</sub>	Photochemical reactions	Lung irritation, systemic effects	Heart failure
SO <sub>2</sub>	Coal burning, industry	Vasoconstriction, platelet aggregation	Arrhythmias

Source: WHO Air Pollution and Health Database

### Indirect effects on cardiac function

In addition to their direct harmful effects, air pollution can worsen pre-existing cardiovascular diseases and cause the emergence of new ones via a number of indirect pathways:

**Systemic inflammation and oxidative stress:** Pollutant inhalation causes pulmonary inflammation, which in turn causes the blood to fill with pro-inflammatory cytokines. Endothelial dysfunction, a crucial step in atherogenesis, may result from this systemic inflammation.

**Autonomic nervous system imbalance:** The autonomic nervous system is susceptible to pollutants, which can change heart rate variability and encourage arrhythmias.

**Pro-thrombotic effects:** Studies have linked exposure to air pollution to elevated blood coagulability, which raises the risk of thrombosis and related cardiovascular events [9,12,13].

### Cardiovascular Risk in Cancer Survivors

The number of cancer survivors is increasing as a result of improvements in cancer therapy that have greatly increased survival rates. However, because of the cardiotoxic effects of several cancer treatments, these people are more likely to acquire cardiovascular diseases (CVDs). The mechanisms of cardiotoxicity

### Carbon monoxide

The incomplete combustion of fuels containing carbon produces CO, an odorless and colorless gas. Because of its strong affinity for hemoglobin, it can create carboxyhemoglobin, which decreases the amount of oxygen that reaches tissues. Myocardial ischemia may result from this hypoxic state, which makes the heart work harder, especially in people who already have cardiovascular disease. A higher risk of arteriosclerosis and other cardiovascular disorders has been associated with long-term exposure to low CO levels [11].

### Ozone

Nitrogen oxides and volatile organic molecules undergo photochemical reactions to produce ground-level ozone. Higher cardiovascular morbidity and death have been linked to O<sub>3</sub> exposure. O<sub>3</sub> may cause inflammation and oxidative stress, which can compromise vasodilation and cause endothelial dysfunction. Furthermore, exposure to O<sub>3</sub> may change how the autonomic nervous system functions, which might lead to arrhythmias and other cardiac irregularities [12]. Common air contaminants and cardiovascular disease are strongly associated, according to numerous research. Table 1 lists the main sources, biological processes, and associated cardiovascular diseases of key pollutants, including PM<sub>2</sub>, NO<sub>2</sub>, O<sub>3</sub>, and SO<sub>2</sub>.

associated with oncology and the long-term cardiac hazards that cancer survivors confront are examined in this section.

### Chemotherapy-induced cardiotoxicity

Despite their effectiveness as chemotherapeutic drugs, anthracyclines, like doxorubicin, are linked to dose-dependent cardiotoxicity. The main process is the production of reactive oxygen species, which causes mitochondrial damage, oxidative stress, and ultimately cardiomyocyte death. Congestive heart failure and dilated cardiomyopathy may follow from this. The risk may appear years after therapy is finished and rises with cumulative dose. Myocardial necrosis and bleeding can result from other chemotherapeutic treatments, such as alkylating agents like cyclophosphamide, which can induce either acute or chronic heart failure. Although less common, antimetabolites and microtubule inhibitors have also been linked to negative cardiac consequences [14].

### Targeted therapies and cardiovascular risk

Heart function may be unintentionally impacted by targeted medicines, which are intended to block certain biochemical processes in cancer cells. For example, vascular problems, such as arterial occlusive events, have been linked to tyrosine kinase inhibitors (TKIs) that target the BCR-ABL fusion protein in

chronic myeloid leukemia. When administered in conjunction with anthracyclines, HER2 inhibitors for HER2-positive breast cancer can cause heart failure and decreases in left ventricular ejection fraction [15].

### Radiation therapy and cardiac complications

Radiation therapy has serious long-term cardiovascular hazards, particularly when it affects the mediastinal region. Conditions include restrictive cardiomyopathy, valvular heart disease, and pericardial constriction can result from radiation's ability to cause inflammation and fibrosis of the myocardial, valves, and pericardium. Another issue is coronary artery disease, where radiation speeds up atherosclerosis and raises the risk of myocardial infarction. After therapy, some side effects could not show up for years or even decades [16]. The long-term cardiotoxic potential of various oncologic therapies continues to be a pressing issue in survivorship care. Table 2 presents a comparative overview of frequently used cancer treatments, their underlying mechanisms of cardiovascular toxicity, and related complications.

### Impacts of Pollution and Oncology

In cardio-oncology, the relationship between environmental pollution and cancer survivability has become a crucial topic of concern. Managing the long-term cardiovascular hazards that cancer survivors experience has emerged as a new concern since oncology discoveries have greatly increased the survival time of cancer patients. The potential of environmental exposures, particularly air pollution, to exacerbate cardiovascular morbidity and death has drawn attention. Pollutants including ozone, nitrogen oxides (NO<sub>x</sub>), and fine particulate matter (PM<sub>2.5</sub>) are known to cause endothelial dysfunction, oxidative stress, and systemic inflammation all of which are important processes in the development of cardiovascular disease (CVD). These side effects might exacerbate the cardiovascular strain in patients already weakened by cancer treatments [10,13].

One known cause of heart disease in the general population is air pollution. However, the negative cardiovascular consequences of pollution can be exacerbated in cancer patients, many of whom have previously had cardiotoxic therapies like radiation and chemotherapy. While thoracic radiation can result in fibrosis and vascular damage, myocardial injury is known to be caused by HER2-targeted drugs and anthracycline-based chemotherapy. There is an increased risk of heart failure, arrhythmias, coronary artery disease, and stroke when these treatment-induced damage are exacerbated by long-term exposure to air pollution. According to recent research, cancer patients who reside in locations with higher PM<sub>2</sub> concentrations have a higher risk of cardiac events than those who live in cleaner surroundings. This suggests that

environmental toxicity and treatment-associated cardiac susceptibility are directly connected [17].

Pollutants such as PM<sub>2.5</sub> can, mechanistically, infiltrate the systemic circulation, enter the lungs, and cause extensive inflammation. This strains an already injured myocardial and speeds up atherosclerosis. Pollutant-induced damage is more severe and more difficult to recover from in cancer patients, whose immune systems and tissue repair systems may be weakened by the disease or its therapies. Pollutants can also interfere with metabolic control by acting as endocrine disruptors. This is especially important for hormone-sensitive tumors like breast and prostate cancer types that are already linked to an elevated risk of cardiovascular disease [18].

The topic of health inequalities adds another level of complexity to this problem. Cancer patients from low-income neighborhoods frequently live in more polluted locations and have less access to high-quality medical treatment. This combination lowers the possibility of early identification and treatment of developing cardiovascular problems in addition to increasing their exposure to detrimental environmental influences. It is even more important to include environmental health evaluations in cancer survival care plans because of these systemic injustices [14,18].

Better patient outcomes in clinical practice can be informed by knowledge of environmental dangers. As part of the cardiovascular risk assessment, doctors who treat cancer patients should take environmental exposure history into account. Simple yet effective interventions might include preventive measures like avoiding outdoor activities during times of high pollution and reducing exposure to contaminated air through indoor filtration. To reduce these synergistic hazards and provide comprehensive treatment for cancer survivors, oncologists, cardiologists, public health authorities, and environmental scientists must work together [19].

### Mechanistic Insights

Through intricate biological processes, it is recognized that air pollution and several cancer treatments might exacerbate cardiovascular illnesses (CVDs). Endothelial dysfunction, inflammation, and oxidative stress are important mechanisms that cumulatively harm cardiovascular health.

**Oxidative stress:** This condition occurs when the body's antioxidant defenses and the generation of reactive oxygen species (ROS) are out of balance. When pollutants like fine particulate matter (PM<sub>2.5</sub>) are inhaled, more reactive oxygen species (ROS) are produced, which damages proteins, DNA, and lipids. Both hypertension and atherosclerosis are exacerbated by this damage. Similarly, the overproduction of ROS caused by cancer treatments like radiation therapy and

**Table 2.** Cancer treatments and cardiovascular complications.

Therapy Type	Mechanism of Cardiotoxicity	Associated Long-term Cardiovascular Risks
Anthracyclin	ROS generation, apoptosis of cardiomyocytes	Dilated cardiomyopathy, heart failure
Trastuzumab	HER2 receptor blockade, mitochondrial dysfunction	Left ventricular dysfunction, heart failure
Chest Radiation	Fibrosis, microvascular damage	Valvular disease, pericarditis, coronary stenosis
VEGF inhibitors	Hypertension, endothelial toxicity	Thromboembolism, heart failure

**Source:** ESC Cardio-Oncology Guidelines, 2022

anthracyclines can increase the risk of heart failure by causing cardiomyocyte damage and apoptosis [12].

**Inflammation:** By triggering immunological responses and generating pro-inflammatory cytokines, exposure to air pollution can cause systemic inflammation. This inflammatory condition speeds up the development of atherosclerotic plaque and encourages endothelial dysfunction. Inflammation brought on by cancer treatments can increase the risk of cardiovascular disease [13].

**Endothelial dysfunction:** By controlling vasodilation and preserving blood fluidity, the endothelium plays a critical role in vascular health. By decreasing nitric oxide (NO) bioavailability, oxidative stress, and inflammation can affect endothelial function by causing vasoconstriction and encouraging thrombosis. This dysfunction is a shared mechanism by which cancer treatments and air pollution both lead to CVDs [6,13].

**Synergistic effects:** The cardiovascular system of cancer patients may suffer additive or even synergistic harm from the combination of environmental contaminants and cardiotoxic treatments. For example, a weakened endothelium brought on by pollution exposure may be more vulnerable to the harmful effects of ROS produced by chemotherapy, which could increase the risk of cardiovascular problems [14]. Developing focused therapies to reduce cardiovascular risks in cancer patients exposed to environmental contaminants requires an understanding of these molecular mechanisms.

## Public Health Implications and Preventive Strategies

A rising public health concern is the relationship between cardiovascular disease, cancer survivorship, and air pollution. A coordinated public health response is essential as there is growing evidence that environmental contaminants increase cardiovascular risk in cancer patients. This comprises all-encompassing approaches that acknowledge environmental exposure as a major factor in determining long-term health outcomes, such as population-based screening, strong policy frameworks, and customized survivorship care models [20].

Programs for population-based screening are essential for identifying at-risk individuals early on. Regular cardiovascular risk assessments must to be incorporated into long-term follow-up procedures for cancer survivors, especially those who have undergone cardiotoxic treatments. Risk stratification can be more accurate if environmental exposure history is taken into account, such as a home's proximity to busy streets or industrial zones. Additionally, non-invasive imaging methods (such as echocardiography or coronary artery calcium scoring) for subclinical cardiovascular disease screening may aid in the early detection of vascular alterations in this high-risk population, allowing for prompt management [21]. Addressing this multidimensional health challenge requires actionable, policy-driven, and clinical-level strategies. Table 3 outlines key interventions, their implementation levels, and anticipated public health outcomes.

**Table 3.** Strategies for cardiovascular protection in cancer survivors.

Strategy	Implementation Level	Target Population	Expected Outcome
HEPA filters at home	Individual	Immunocompromised, cancer survivors	Reduced exposure to indoor PM <sub>2.5</sub>
Air quality index (AQI) alerts	Community	Urban residents, elderly	Behavioral changes on high pollution days
Stricter vehicle emission norms	Policy	General public	Lower ambient air pollution levels
Annual CV risk assessments	Clinical	Post-therapy cancer patients	Early detection and intervention
Green infrastructure planning	Urban policy	Highrisk zones	Long-term reduction in localized pollution

In order to reduce environmental threats, public policy is essential. To provide the public with easily available data, governments must invest in real-time air monitoring devices and implement strict air quality standards. Pollutant levels can be considerably decreased by implementing low-emission zones, encouraging green urban infrastructure, and switching to cleaner energy sources, which will benefit vulnerable groups like cancer survivors. Healthcare professionals should be prepared with guidelines that integrate pollution exposure into clinical decision-making and informed on environmental risk factors. In order to create evidence-based policy recommendations, interdisciplinary cooperation between environmental scientists, public health officials, and healthcare practitioners is crucial [9,21,22].

Environmental health must become a fundamental part of survivorship care approaches. This includes patient education in addition to clinical surveillance. Cancer survivors should receive advice on how to reduce their exposure to pollution, such as utilizing HEPA filters indoors, keeping an eye on local air quality indices, and staying indoors during peak pollution

hours. To improve patient involvement and adherence to preventive measures, telemedicine and mobile health apps can be used to deliver tailored notifications and health coaching [23].

In the end, it is impossible to address cancer survivors' long-term cardiovascular health in a vacuum, divorced from their surroundings. We may take a more comprehensive approach that better serves this expanding patient group and lessens the burden of pollution-related cardiovascular disease on society by including environmental risk factors into public health policy, screening, and care delivery models [24].

## Future Directions and Research Gaps

There are still a lot of unanswered questions despite mounting evidence that air pollution has a negative impact on cardiovascular outcomes in cancer survivors. Emerging research needs to go closer into the molecular underpinnings of pollutant-induced cardiotoxicity, especially in oncology contexts. Multi-omics techniques, such as transcriptomics, metabolomics, and genomes, can provide new biomarkers that



forecast a person's vulnerability to environmental exposures and treatment-induced cardiovascular disease. Furthermore, well-designed longitudinal cohort studies that track cancer survivors over decades are desperately needed in order to evaluate the timing of cardiovascular events and the cumulative effects of exposure. Causal inference is limited by the cross-sectional nature of current data. Pediatric cancer survivors are also underrepresented because the majority of studies have concentrated on adult populations. Health disparities must also be given top priority in research, which looks into how socioeconomic and geographic factors affect exposure levels and results. In the age of environmental and oncologic overlap, closing these gaps will open the door for precision prevention initiatives, individualized survivorship treatment, and policy changes that take into account the intricate, multifaceted nature of cardiovascular risk [7,25].

## Conclusions

The relationship between cardiovascular health, cancer survivorship, and air pollution is a pressing yet little-known public health issue. As cancer treatments increase survival, focus needs to move to reducing long-term risks, especially cardiovascular issues caused by environmental factors. An integrated strategy that incorporates clinical vigilance, policy action, and individualized care is necessary due to the combined effects of pollution and cardiotoxicity connected to oncology. We can lessen the worldwide burden of cardiovascular disease, enhance survivorship quality, and better protect vulnerable people by acknowledging environmental exposure as a modifiable risk factor. To fill in the gaps and direct evidence-based solutions, future research and policy must collaborate.

## Disclosure statement

No potential conflict of interest was reported by the authors.

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