

Systematic Review

Impact of Climate Change on Cancer: Escalating Risks and Urgent Concerns

Alaa Fadhil Alwan ^{1,a}, Salah Hashim Al-Zuhairy ^{1,b}, Said Nabil ^{2,3,c}, Shahbaa Alshebli ^{4,d}, Anas Hamad ^{5,6,e}, Mohamed Abdulroaf Elsisi ^{7,f}, Sara Essam Bauomy Ibrahim ^{8,g}, Muhammed Hassan Nasr ^{8,h}, Saba Mahdi Jasim ^{9,i*}

¹ The National Center of Hematology, Mustansiriyah University, Baghdad, Iraq.

² Department of Pharmacy Practice, College of Pharmacy, Gulf Medical University, Ajman, United Arab Emirates.

³ Department of Clinical Pharmacy, Thumbay University Hospital, Ajman, United Arab Emirates.

⁴ Department of Pharmacology and Therapeutics, College of Medicine and Health Sciences, United Arab Emirates University, Al Ain, United Arab Emirates.

⁵ Pharmacy Department, National Center for Cancer Care & Research, Hamad Medical Corporation, Doha, Qatar.

⁶ College of Pharmacy, QU Health Sector, Qatar University, Doha, Qatar.

⁷ Hayat National Hospital - Al-Madinah Al-Munawwarah, Saudi Arabia.

⁸ Department of Clinical Pharmacy, Al Rayan National College of Health Sciences, Al Rayan National Colleges, Al Madinah Al Munawwarah, Saudi Arabia

⁹ Oncology Teaching Hospital, Baghdad, Iraq.

E-mail: ala_sh73@uomustansiriyah.edu.iq ^a; salahalzuhairy@uomustansiriyah.edu.iq ^b; 700036435@uaeu.ac.ae ^c; 700048313@uaeu.ac.ae ^d; ahamad6@hamad.qa ^e; tccdr242011@gmail.com ^f; Se.ibrahim@amc.edu.sa ^g; Mh.nasr@amc.edu.sa ^h; sabamj.onco@gmail.com ^{i,*}

Received: 17 October 2025 | Revised: 06 January 2026 | Accepted: 28 March 2026 | Published: 13 May 2026

Abstract

The present review examines the complex relationship between climate change and cancer, focusing on how environmental changes may influence cancer risk and outcomes. Despite growing evidence, research on the climate–cancer nexus remains heterogeneous and geographically limited, highlighting the need for more robust data from diverse populations. This review aims to improve understanding among oncology professionals of the potential role of climate change in cancer incidence and to support the development of comprehensive strategies that integrate the broader context of cancer prevention in a rapidly changing world. A comprehensive search of the PubMed database was conducted to identify English-language scholarly literature addressing climate change and various solid cancers in human studies. Search terms included “air pollution,” “ozone depletion,” “ultraviolet radiation,” “global warming,” and “different types of solid cancer.” A detailed scoping review of the available literature was undertaken to examine the multiple ways in which climate change may contribute to cancer risk. The findings indicate associations between factors such as increased exposure to ultraviolet radiation, air pollution, and disruptions to food and water supplies and the incidence of several major solid cancers, drawing on evidence synthesised from multiple epidemiological studies. The review also examines shared pathways, underlying mechanisms, and practical implications across cancer types. By bringing together current evidence on the relationship between climate change and solid cancer incidence, this article identifies important considerations for future research and public health strategies. Although individual efforts to reduce exposure to modifiable risk factors are underway, further research is needed to determine which measures are most effective in minimizing their adverse effects on health.

Keywords: Cancer Incidence; Cancer Mortality; Climate Change; Risk Factors; Treatment Strategies.

* Correspondence Author

Copyright: © 2026 by the authors. Licensee Scientific Steps International Publishing Services, Dubai, UAE.

This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

Cite This Article: Alaa Fadhil Alwan, Salah Hashim Al-Zuhairy, Said Nabil, Shahbaa Alshebli, Anas Hamad, Mohamed Abdulroaf Elsisi, Sara Essam Bauomy Ibrahim, Muhammed Hassan Nasr, Saba Mahdi Jasim (2026).

Impact of Climate Change on Cancer: Escalating Risks and Urgent Concerns. *Middle Eastern Cancer and Oncology Journal*, 2(2), 33–53. <https://doi.org/10.61706/MECOJ160214>

Introduction

Cancer continues to represent a primary cause of mortality and disability on a global scale. While classical risk factors (tobacco, diet, infections, occupational exposures) have been thoroughly delineated, climate-linked hazards are now recognised to intersect with many of these pathways. Temperature increases, shifts in atmospheric chemistry, lengthened wildfire seasons, extreme precipitation and flooding, and volatile food and water security are all associated with changes in the levels, mixtures and distribution of carcinogenic exposures (Nogueira et al., 2020; Ray & Ming, 2020; P. Yu et al., 2023). These changes in turn impact health systems, which are under pressure to deliver screening and treatment. In addition to direct environmental exposure, climate change exerts an influence on social determinants of cancer risk, including forced migration, urban crowding, and shifting occupational exposures. These factors contribute to the shaping of both incidence and outcomes (Schwerdtle et al., 2018).

The phenomenon of air pollution, which has already been classified by the International Agency for Research on Cancer (IARC) as carcinogenic to humans, serves to illustrate this convergence. Fossil-fuel combustion, wildfires, and secondary aerosol formation have been identified as key drivers of exposure to fine particulate matter and copollutants. These have been associated with an increased risk of lung cancer, and increasingly, other sites (Nogueira et al., 2020; Ray & Ming, 2020; P. Yu et al., 2023). The dynamics of ozone and the interactions between the stratosphere and the troposphere have been demonstrated to influence surface ultraviolet (UV) radiation and, by extension, the risk of developing skin cancer. It is important to note that heat waves and disasters have the potential to disrupt oncology care, for instance in terms of access to radiotherapy and the logistics of chemotherapy. This disruption can lead to a deterioration in the survival rates of vulnerable populations. In the context of warming conditions, food and water insecurity, including mycotoxins (e.g. aflatoxins) and cyanotoxins, represent additional pathways that have been demonstrated to be relevant to gastrointestinal and hepatic malignancies (Kehm et al., 2025). The mechanisms through which climate change influences cancer can be broadly divided into two categories: direct and indirect pathways. Direct pathways pertain to environmental exposures that have been demonstrated to possess carcinogenic potential, including fine particulate matter, ultraviolet radiation, and aflatoxins. In contrast, indirect pathways encompass the social and systemic consequences of climate instability, such as healthcare disruption, forced migration, and delayed cancer screening. The recognition of these distinctions

is of paramount importance, as it enhances causal interpretation and guides targeted prevention efforts.

Notwithstanding the burgeoning corpus of literature on the subject, oncology guidance has not yet fully integrated climate-health evidence into prevention and service planning. This review synthesises recent human studies on climate-relevant exposures and solid cancers, highlighting plausible mechanisms and outlining practice and policy implications for cancer control in a warming world. Notwithstanding the heightened level of research activity, considerable knowledge gaps persist. A paucity of studies has systematically examined cancers beyond those of the lung and skin, and data from low- and middle-income regions—where climate vulnerability and environmental exposures are highest—remain sparse. Addressing these geographic and cancer-site imbalances is essential to building an equitable global evidence base. The present review seeks to systematically synthesize evidence linking climate-related exposures to cancer incidence, progression, and outcomes through an integrated framework that distinguishes environmental, biological, and health-system pathways. The conceptual framework underpinning this review posits that climate change exerts its influence on cancer through two distinct pathways: (i) direct pathways, encompassing exposure to carcinogens such as air pollution and ultraviolet radiation; and (ii) indirect pathways, including food and water contamination, occupational exposures, and disruption of oncology care during extreme events. The objective of this study is to provide a comprehensive understanding of how climate hazards collectively reshape global cancer risks by mapping these interrelated mechanisms.

Methods

Design and Registration

A structured scoping review was conducted in accordance with PRISMA-aligned best practices for transparent identification and synthesis of evidence on climate-relevant exposures and solid tumours (the protocol for which is available upon request). The PRISMA flow chart can be found in Figure 1.

Sources and Search

A comprehensive search was conducted on PubMed, SCOPUS, and Web of Science for English-language records from 2010 to 2024. This search employed a combination of controlled vocabulary and keywords that amalgamated cancer terminology (e.g., cancer, neoplasm, specific sites) with climate-related exposures (e.g., particulate matter, air pollution, wildfire smoke, ultraviolet, ozone, heat, global warming, drought, water/food contamination). To

capture additional articles, a hand-search was conducted of reference lists of key reviews and cohort studies.

Eligibility Criteria

The following criteria must be met in order to be eligible. The present study incorporated human studies evaluating solid cancers in relation to at least one climate-relevant exposure or event (as previously defined). Eligible designs encompassed cohort, case-

control, cross-sectional studies, meta-analyses, and narrative/systematic reviews reporting incidence, mortality, progression, or care disruption. In the course of the present study, the following exclusions were made: non-English articles, animal/mechanistic studies devoid of human data, editorials lacking primary/secondary data, and abstracts not accompanied by the full text.

PRISMA (Simplified) — Climate-Relevant Exposures & Solid Tumors

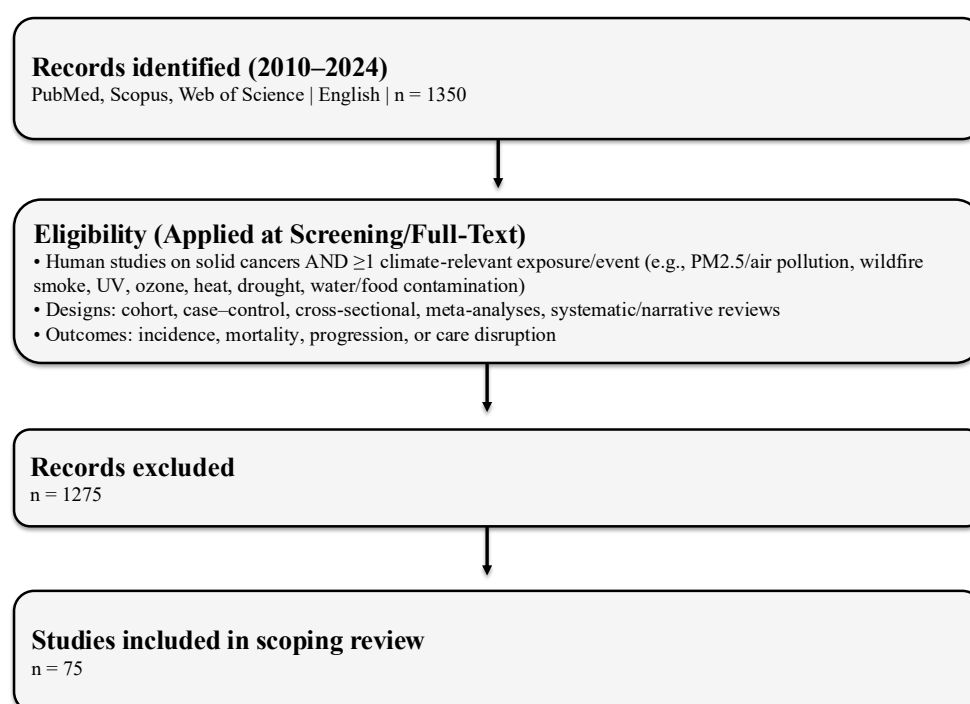


Figure 1. PRISMA Flow Diagram of Study Selection on Climate-Relevant Exposures and Solid Tumours.

Study Selection and Appraisal

The initial screening of titles and abstracts was conducted by two reviewers, who subsequently undertook a full-text assessment against the predetermined eligibility criteria. Discrepancies were resolved through consensus. In instances where relevant, the following domains were noted: exposure assessment methods (e.g. land-use regression, satellite-derived PM_{2.5}), outcome ascertainment, confounder control, and risk-of-bias domains (selection, exposure misclassification, confounding, and outcome misclassification).

Synthesis

Given the heterogeneity in exposures, populations, and endpoints, the findings were narratively synthesised by cancer site and exposure domain (air pollution, UV/ozone, heat and extremes, food/water contaminants, care disruption). In instances where

meta-analytic estimates were available, the direction and strength of the observed association were reported, as opposed to the re-pooling of data. Although quantitative synthesis (meta-analysis) was initially considered, substantial heterogeneity across study designs, exposure metrics, and cancer outcomes precluded statistical pooling. Consequently, a narrative synthesis approach was adopted in order to integrate findings by exposure type and cancer site, in accordance with scoping review methodology. This approach is in accordance with PRISMA-ScR guidance for synthesizing evidence in emerging interdisciplinary fields where there is a paucity of standardized effect estimates.

The Global Climate Change

Global climate change, characterised by substantial changes in average weather patterns and an increase in extreme weather events (Ray & Ming, 2020; P. Yu et al., 2023), is having a profound impact on the

planet. This phenomenon has been extensively documented and is primarily driven by human activities. The European Environment Agency has reported a steady rise in global sea levels, averaging 1.8 mm per year since 1961, alongside a concerning 2.7% per decade decrease in Arctic Ocean ice coverage (Blockley et al., 2023). According to Ray and Ming (2020), these changes have contributed to the retreat of mountain glaciers, increased ocean acidity, and a higher frequency of natural disasters. The Intergovernmental Panel on Climate Change (IPCC) has warned that extreme weather events, including heatwaves, floods, and droughts, are expected to occur with increasing frequency throughout the 21st century (Blockley et al., 2023). As environmental challenges intensify, achieving sustainable development becomes increasingly complex. A key aspect of this challenge is understanding the effects of climate change on human health, particularly in relation to cancer research and prevention (Ray & Ming, 2020).

The Global Cancer Burden and Climate Change

In recent decades, cancer has emerged as a leading cause of premature death worldwide, surpassing cardiovascular disease in some contexts and representing a major global health concern. Many cancers that were once considered inevitably fatal can now be prevented in some cases or treated more effectively (P. Yu et al., 2023). This progress can be attributed to advances in early detection, more effective treatments, and improved control of cancer risk factors. Despite these advances, disparities in exposure to risk factors and access to healthcare continue to contribute to differences in cancer incidence and mortality rates worldwide (P. Yu et al., 2023).

Nevertheless, climate change poses a critical yet frequently overlooked challenge to ongoing efforts in cancer prevention and control. It affects two distinct but interconnected areas: exposure to cancer risk factors and access to cancer care (Nogueira et al., 2020; Schiller et al., 2020; P. Yu et al., 2023). For example, extreme weather events, such as hurricanes and wildfires, can increase exposure to toxic substances, potentially contributing to greater cancer risks (Jones et al., 2022). Furthermore, the effects of climate change on cancer outcomes, including survival, are becoming an increasingly important concern (Nogueira et al., 2020; Schiller et al., 2020; P. Yu et al., 2023). Extreme weather events can disrupt cancer treatment facilities and limit patients' access to care (Hiatt & Beyeler, 2020). Consequently, strengthening disaster preparedness in cancer treatment centres and addressing the healthcare sector's contribution to global warming are important priorities. Notably, many strategies designed to mitigate climate change can also reduce exposure to carcinogens, potentially providing dual benefits for

climate action and cancer prevention (Hiatt & Beyeler, 2020).

Increased Exposure to Carcinogens Due to Climate Change

Anthropogenic climate change may increase cancer risk by increasing exposure to established carcinogens through various environmental pathways, including air, water, soil, food, and ultraviolet (UV) radiation. Outdoor air pollution, particularly its fine particulate matter component (PM_{2.5}), has been identified as carcinogenic to humans, with established evidence linking it to lung cancer (PRESS RELEASE NO. 221 IARC: Outdoor Air Pollution a Leading Environmental Cause of Cancer Deaths, 2013; Turner et al., 2020). Extreme weather events can cause acute increases in exposure to pollutants. For example, during Hurricane Harvey in 2017, petrochemical facilities reportedly released more than one million pounds of air pollutants, while 13 nearby Superfund sites were flooded or potentially damaged (US EPA, n.d.). Dioxins illustrate the long-term persistence of some environmental contaminants, with a biological half-life in humans of approximately 7–11 years and estimated half-lives in soil of around 9–15 years in surface soil and 25–100 years in subsurface soil (Dioxins, n.d.). In quantitative terms, ambient PM_{2.5} was estimated to account for approximately 14.1% (95% UI 9.8–18.7) of global lung cancer deaths in 2017, highlighting a substantial and potentially preventable burden that may be affected by climate-related changes in exposure (Turner et al., 2020). These impacts are not distributed equitably. In the areas affected by Hurricane Harvey, flooding and toxic incidents disproportionately affected neighbourhoods with larger non-Hispanic Black populations and greater socioeconomic deprivation. This pattern underscores the importance of environmental justice (Blockley et al., 2023; Mufson, n.d.; PRESS RELEASE N° 221 IARC: Outdoor Air Pollution a Leading Environmental Cause of Cancer Deaths, 2013; Turner et al., 2020; US EPA, n.d.).

Furthermore, the expansion of wildfire seasons, together with increases in fire frequency and severity, has contributed to the release of substantial quantities of air pollutants, including particulate matter and other potentially carcinogenic substances. These emissions can affect air quality over extensive areas, as illustrated in 2018, when smoke from California's wildfires affected air quality across the continent (Claire, 2025; Mufson, n.d.; PRESS RELEASE N° 221 IARC: Outdoor Air Pollution a Leading Environmental Cause of Cancer Deaths, 2013; Turner et al., 2020).

Climate change may also exacerbate air pollution through several interconnected processes, including changes in atmospheric conditions and the continued combustion of fossil fuels, which releases greenhouse gases and air pollutants. The International Agency for

Research on Cancer (IARC) has classified outdoor air pollution as carcinogenic to humans (Group 1), underscoring its significance as a public health concern. Both indoor and outdoor air pollution present important health risks, including cancer risks. Millions of people are exposed to indoor air pollution from the combustion of biomass fuels and coal, which can further contribute to the burden of harmful exposures (Chakraborty et al., 2019; Claire, 2025; Dioxins, n.d.; Lieberman-Cribbin et al., 2021; US EPA, n.d.).

In addition, rising temperatures associated with climate change may pose particular challenges for people with cancer. Some patients may be more vulnerable to extreme heat because of impaired thermoregulation, treatment-related effects, or other health-related factors. Increasing occupational heat exposure is also a concern, although the relationship between heat exposure and specific cancers, including breast cancer, requires careful evaluation of the available evidence. Changes in stratospheric ozone levels can increase exposure to ultraviolet (UV) radiation, which is an established risk factor for melanoma and other skin cancers. Furthermore, melanoma survivors have an increased risk of developing second primary malignancies, including prostate cancer (Claire, 2025; Mufson, n.d.; US EPA, n.d.).

Climate change also raises concerns about water quality. Conditions associated with warmer temperatures may favour the proliferation of toxin-producing cyanobacteria, increasing the potential for human exposure to cyanotoxins through contaminated water supplies. Some cyanotoxins have been investigated for possible associations with liver and colorectal cancers, although the strength of evidence varies by toxin and cancer type. In addition, climate-related changes in growing conditions and food storage may increase the risk of aflatoxin contamination in food crops. Aflatoxins are potent carcinogens produced by certain fungi, and exposure to them is an established risk factor for liver cancer (Chakraborty et al., 2019; Dioxins, n.d.; PRESS RELEASE N° 221 IARC: Outdoor Air Pollution a Leading Environmental Cause of Cancer Deaths, 2013; Lieberman-Cribbin et al., 2021; Mufson, n.d.; Turner et al., 2020; US EPA, n.d.).

These trends highlight the need for comprehensive strategies to address the intersection of climate change and cancer. A clearer understanding of the pathways linking climate-related environmental changes to carcinogen exposure and cancer risk is essential for developing effective prevention and adaptation strategies to address this growing public health concern.

As Healthcare Professionals, What Can We Do to Address This Critical Situation?

Healthcare professionals, including doctors and other medical staff, play a pivotal role in global efforts to address climate change, drawing on their credibility as trusted sources of information. By using this trust to communicate health risks and advocate for effective action, healthcare professionals can make important contributions to protecting patients' health and well-being. This includes supporting policies that mitigate the adverse effects of climate change on human health (Hiatt & Beyeler, 2020; P. Yu et al., 2023). Healthcare organisations can contribute to a healthier environment and population by supporting measures that reduce the environmental impact of society and the healthcare sector. Promoting sustainable practices and environmentally responsible initiatives is important for climate change mitigation and can also provide broader benefits for public health and well-being (Hiatt & Beyeler, 2020).

Healthcare professionals should also participate proactively in preparedness efforts for emerging infectious diseases and other health threats that may be influenced by climate change. They can contribute by advocating for comprehensive preparedness strategies and supporting research aimed at understanding and addressing these evolving threats, thereby strengthening community resilience (Chakraborty et al., 2019; Hiatt & Beyeler, 2020; Lieberman-Cribbin et al., 2021).

Recognizing the relationship between climate change and human health, including the growing concern about its potential links with cancer, is another important aspect of healthcare advocacy. Recent studies suggest that climate change may indirectly influence cancer risk through environmental and lifestyle changes. These potential relationships highlight the need for further research to clarify the underlying pathways and better understand the extent of these effects (Hiatt & Beyeler, 2020; Lieberman-Cribbin et al., 2021).

Active involvement in research and education at the intersection of climate change and health is also essential. Generating and sharing knowledge in this field can help healthcare professionals raise awareness, inform communities, and contribute to policy development. Education is fundamental to these efforts, as it helps build a clearer understanding of the health risks associated with climate change and the strategies needed for both mitigation and adaptation (Hiatt & Beyeler, 2020; Lieberman-Cribbin et al., 2021). Detailed evidence is presented in Table 1, while the pathways, underlying mechanisms, and practical implications synthesised in this review are summarized in Table 2.

Table 1. Human Studies on Climate-Relevant Exposures and Cancer

Author (Year)	Country/Setting	Design & Sample Size	Exposure(s)	Cancer Outcome(s)	Key Findings	Notes/Limits
(Hill et al., 2023)	Multinational	Experimental + integrative (Nature)	PM _{2.5}	Lung adenocarcinoma	PM _{2.5} promotes expansion of oncogenic clones in airway epithelium	Mechanistic focus; not a population risk estimate
(Guo et al., 2024)	Southern China	Large cohort	Air pollution (long-term)	Lung cancer incidence/admissions	Long-term pollution positively associated with lung cancer metrics	Exposure misclassification possible
(Ma et al., 2023)	Osaka, Japan	Time-series	Short-term PM and gases	Lung-cancer mortality	Daily pollution spikes associated with higher mortality	Short-term associations
(Korsiak et al., 2022)	Canada	Nationwide cohort	Wildfire exposure (long-term)	Overall & site-specific incidence	Wildfire smoke linked to higher overall and lung cancer incidence	Exposure modeled; residual confounding
(Hassanipour et al., 2023)	Global	Systematic review & meta-analysis	Ambient pollutants	CNS/brain tumors	Positive pooled associations for CNS tumors	Heterogeneous designs/exposures
(Pagano et al., 2023)	Narrative/mechanistic	Review	Air pollution mixtures	Brain tumorigenesis	Mechanistic pathways: oxidative stress, BBB disruption	Narrative synthesis
(Ruszkiewicz et al., 2019)	Global	Review	Climate-linked toxicants	Brain diseases incl. tumors	Summarizes neuro-inflammatory/oxidative mechanisms	Not tumor-specific meta-analysis
(Wei et al., 2023)	USA	Large cohort (older adults)	PM _{2.5} + NO ₂ (10-yr)	Primary cancer incidence	Additive effects of PM _{2.5} and NO ₂	All-site incidence; site-specific detail limited
(Parent et al., 2013)	Montreal, Canada	Case-control (~1,900+)	NO ₂ (traffic)	Prostate cancer	Higher NO ₂ linked with increased prostate cancer risk	Urban exposure model; confounding possible
(Youogo et al., 2022)	Canada	Case-control (1,420/1,424)	PM _{2.5} & NO ₂	Prostate cancer	Long-term PM _{2.5} /NO ₂ associated with prostate cancer risk	Observational; exposure modeling
(Chen et al., 2022)	Europe	Pooled cohorts	Ambient air pollution	Bladder cancer incidence	Pollution associated with higher bladder-cancer incidence	Low-level exposure focus
(Sakhvidi et al., 2020)	Global	Systematic review	Air pollution	Bladder, kidney, urinary tract cancers	Positive associations; calls for refined exposure metrics	Heterogeneity across studies
(Hvidtfeldt et al., 2022)	Europe	Cohort	Ambient air pollution	Kidney cancer	Associations observed even at low levels	Potential residual confounding
(Nagel et al., 2018)	Europe	6-cohort study	PM _{2.5} /NO ₂	Stomach & aerodigestive cancers	Positive associations across some sites	Exposure models; site heterogeneity
(Jenwitheesuk et al., 2020)	Thailand	Ecologic/time-series	Accumulated air pollution	Colon cancer incidence	Pollution associated with increased colon cancer incidence	Ecologic design
(Chu et al., 2021)	China	Prospective cohort (gene interaction)	PM _{2.5} + genetics	Colorectal cancer	Gene–environment interactions observed	Needs replication
(Ku et al., 2021)	Taiwan	Cohort	PM _{2.5}	CRC pathway	PM _{2.5} linked to elevated fecal Hb & CRC risk	Intermediate outcome

Author (Year)	Country/Setting	Design & Sample Size	Exposure(s)	Cancer Outcome(s)	Key Findings	Notes/Limits
(So et al., 2021)	Europe	Multi-cohort	Air pollution	Liver cancer incidence	Long-term pollution associated with liver-cancer risk	Residential exposure
(Gan et al., 2023)	Systematic review	Review/meta-narrative	Air pollution	Liver cancer	Positive associations with incidence/mortality	Heterogeneity noted
(Scheurer et al., 2014)	USA	Cross-sectional	Traffic HAPs	Cervical dysplasia	Traffic pollutants associated with dysplasia	Cross-sectional
(Hu et al., 2023)	China	Ecologic panel	Air pollution	Breast & cervical cancer prevalence	Positive population-level associations	Ecologic design
(Dehghani et al., 2022)	Global	Review	Air pollution	Ovarian cancer	Positive associations with ovarian cancer	Narrative; effect sizes vary
(Yin et al., 2020)	Global	Narrative review	Nitrates/nitrites	Gastric cancer	Nitrate exposure linked to gastric cancer risk	Mixed designs
(Dehghani et al., 2021)	Iran	Ecologic/cohort	Air pollution	Thyroid cancer	Higher pollution linked with thyroid-cancer incidence	Exposure limits
(He et al., 2022)	Hunan, China	Population cohort	Ozone	Thyroid nodules	Ozone associated with thyroid nodules	Precursor outcome
(Parker, 2021)	Global	Review	UV + pollutants	Skin cancers	UV remains dominant; pollution may potentiate risk	Narrative synthesis

Table 2. Climate-Related Exposures and Their Links to Cancer: Mechanisms and Implications

Climate-Linked Exposure / Hazard	Associated Cancer(s)	Evidence Strength*	Mechanisms / Pathways	Implications for Practice & Policy
PM _{2.5} , NO ₂ (traffic/industrial, wildfires)	Lung, prostate, colorectal, liver, bladder, kidney, brain, thyroid, oral	Strong (multiple cohorts + meta-analyses)	Oxidative stress, inflammation, endocrine disruption, DNA damage, oncogenic clone promotion	Emission reduction, clean-air shelters, occupational protections, registry linkage with air-quality data
Wildfire smoke	Lung, brain, oral	Moderate (population cohorts; mechanistic plausibility)	Complex mixtures (PAHs, ultrafines), systemic inflammation, neurotoxicity, disruption of care	Smoke-response plans, respirators, continuity of screening and oncology services during wildfire events
UV radiation (ozone depletion, behavior changes)	Skin cancers (melanoma, keratinocyte cancers), lip cancer	Strong (decades of epidemiology + mechanistic clarity)	Direct DNA damage, oxidative stress, immune modulation	UV alerts, occupational protections, sunscreen/protective clothing guidance
Ozone (surface-level)	Thyroid nodules/cancer (emerging evidence)	Low–moderate (ecologic/cohort, precursor endpoints)	Endocrine disruption, oxidative stress	Strengthen air-quality policies, integrate thyroid risk surveillance
Nitrates/nitrites (climate-driven water contamination)	Gastric, thyroid	Moderate (ecologic, cohort, meta)	Nitrosation → N-nitroso compounds; iodide competition in thyroid	Monitor water quality, regulate agriculture runoff, safe drinking-water interventions
Aflatoxin & cyanotoxins (warming, drought, floods)	Liver, colorectal	Strong for aflatoxin–HCC; moderate for cyanotoxins–CRC	DNA adducts, hepatotoxicity, metabolic dysregulation, microbiome disruption	Food-safety systems (drying/storage), toxin surveillance, nutrition policies
Extreme heat / heatwaves	Breast, occupational cancers; service disruption across all	Moderate (occupational & ecologic studies)	Immune suppression, hormone pathway modulation, care delays	Occupational protections, heat emergency oncology service planning

Climate-Linked Exposure / Hazard	Associated Cancer(s)	Evidence Strength*	Mechanisms / Pathways	Implications for Practice & Policy
Care disruption (floods, storms, power loss)	All cancers (treatment delays: radiotherapy, surgery, chemotherapy, screening, vaccination)	High (consistent reports across settings)	Interrupted treatment → later-stage presentation, worse survival	Health-system resilience (backup power, mobile units, flexible scheduling)
Persistent organic pollutants, pesticides, EDCs	Breast, prostate, ovarian	Moderate (epidemiology + mechanistic support)	Estrogenic/inflammatory pathways, endocrine disruption	Regulation of EDCs, occupational/environmental protections

Breast Cancer and Climate Change

Breast cancer is the most frequently diagnosed malignancy among women worldwide. Its aetiology is multifactorial, involving interactions between genetic susceptibility, hormonal and reproductive factors, and environmental exposures (Hiatt & Beyeler, 2023). Susceptibility varies throughout the life course. Prenatal development, puberty, pregnancy and lactation, and the menopausal transition are important windows during which breast tissue may be particularly vulnerable to endocrine-active and inflammatory exposures that could influence long-term cancer risk (Terry et al., 2019). Persistent organic pollutants (POPs), pesticides, and endocrine-disrupting chemicals may also influence breast cancer risk through oestrogenic and inflammatory pathways. Climate-related changes may alter the distribution or mobilisation of some of these substances, potentially changing patterns of exposure (Ennour-Idrissi et al., 2019).

Climate-sensitive hazards may influence both the initiation and progression of breast cancer. Combustion-related mixtures, including fine particulate matter (PM_{2.5}) and polycyclic aromatic hydrocarbons (PAHs) from traffic, industrial activities, and wildfires, have been associated with oxidative stress, systemic inflammation, and endocrine activity relevant to breast carcinogenesis (Cruz et al., 2018; Hinchliffe et al., 2021; Rodgers et al., 2018). Extreme heat and disaster events may also adversely affect dietary quality and increase exposure to hazardous substances in household and occupational settings. In addition, such events can disrupt screening and treatment pathways, potentially exacerbating existing disparities in incidence, stage at diagnosis, and health outcomes. These stressors do not affect all populations equally and may widen disparities among communities exposed to climate-related hazards (Hiatt & Beyeler, 2023; P. Yu et al., 2023).

Geographic patterns provide further grounds for investigating the environmental contribution to breast cancer risk. Ecological analyses in Iran, for example, have identified spatial gradients that may be associated with aridity, solar exposure, and co-occurring air pollution mixtures. These findings provide a basis for

further investigation and support consideration of climate and air-quality indicators in breast cancer surveillance and prevention programmes (Maryanaji, 2020).

The implications for practice and policy can be considered at three levels. Primary prevention should focus on reducing emissions and controlling combustion sources that generate PAHs and other toxic substances. Secondary prevention should include measures to protect workers and communities from heat and wildfire smoke during extreme events. Tertiary prevention should focus on ensuring that healthcare systems can maintain screening, timely pathology services, and oncology care during climate-related emergencies, with health equity as a central objective.

Lung Cancer and Climate Change

Lung cancer is the leading cause of cancer-related mortality worldwide. Although tobacco remains the dominant risk factor, climate-sensitive exposures, particularly combustion-related air pollution and wildfire smoke, are increasingly being investigated for their roles in carcinogenesis and disease progression. Multi-pollutant mixtures containing fine particulate matter (PM_{2.5}) and nitrogen oxides have been associated with systemic inflammation, oxidative stress, and tissue remodelling, processes that may contribute to malignant transformation and tumour growth. Mechanistic research suggests that fine particles may promote the expansion of oncogenic clones in susceptible airway epithelium, providing a biologically plausible link between episodic or chronic exposure to air pollution and lung tumour development (64). Research by Hill et al. (2023), published in *Nature*, further demonstrated that fine particulate matter (PM_{2.5}) may promote the expansion of oncogenic clones in susceptible airway epithelium, providing a potential mechanistic link between air pollution and lung adenocarcinoma, including in non-smokers (Hill et al., 2023).

From an epidemiological perspective, analyses of long-term cohorts have identified associations between exposure to PM_{2.5} and nitrogen dioxide (NO₂) and increased lung cancer incidence and mortality, including in settings where pollutant concentrations are

close to or below previously established regulatory limits (Hill et al., 2023; Rodgers et al., 2018). Studies involving large cohorts of older adults have also reported an additive effect of long-term exposure to PM_{2.5} and NO₂ on the incidence of primary cancer. These findings raise concerns about the combined effects of pollutant exposure under warming conditions that may favour the formation of photochemical smog (Fernandez, n.d.). Short-term increases in particulate matter and gaseous air pollutants have also been associated with lung cancer mortality (Nogueira et al., 2020). This association suggests that acute increases in pollution may be linked to poorer outcomes among people living with cancer, although the nature and extent of this relationship require further investigation.

Climate change is contributing to longer wildfire seasons and may increase the frequency of severe smoke events. A population-based study in Canada reported an association between long-term exposure to wildfires and increased cancer incidence, with a particular association observed for lung cancer (Guo et al., 2024). In addition to potential direct carcinogenic effects, climate-related extreme events, including heatwaves, wildfires, and floods, can disrupt low-dose computed tomography (CT) screening, diagnostic pathways, and treatment logistics. Such disruptions may delay access to stage-appropriate care, particularly for people living in highly exposed or resource-constrained communities (Hill et al., 2023).

The implications for practice include three key priorities. First, primary prevention should focus on rapidly reducing emissions from combustion sources. Second, smoke and smog response plans should prioritise protection for vulnerable groups through measures such as respirators, clean-air shelters, and remote triage. Third, healthcare systems should establish service-continuity plans, including backup power for radiology and infusion services and flexible scheduling during smoke or heat alerts, to minimise interruptions to cancer care during climate-related emergencies (Guo et al., 2024; Hill et al., 2023).

Brain Cancer and Climate Change

Primary brain tumours comprise a diverse group of neoplasms with different histological characteristics and aetiologies. In addition to well-established risk factors, such as ionising radiation and certain genetic syndromes, climate-sensitive exposures, particularly complex air pollution mixtures and wildfire smoke, may influence disease risk and outcomes through mechanisms involving neuroinflammation, oxidative stress, and dysfunction of the blood–brain barrier (BBB) (Hassanipour et al., 2023; Pagano et al., 2023; Ruszkiewicz et al., 2019). These mixtures, which include fine particulate matter and reactive gases released by traffic, industrial activities, and wildfires, have been associated with chronic inflammatory

signalling and redox imbalance in neural tissues. Research also suggests that exposure to elevated temperatures and prolonged exposure to particulate pollutants may compromise the integrity of the BBB, potentially exacerbating neuroinflammation and facilitating the entry of toxic substances into the central nervous system (CNS) (Hassanipour et al., 2023; Pagano et al., 2023; Ruszkiewicz et al., 2019).

Evidence from population-based studies is growing, although the findings remain heterogeneous. A recent systematic review and meta-analysis reported positive associations between several ambient air pollutants and central nervous system (CNS) tumours, while noting variability in study designs and exposure assessment methods (Hassanipour et al., 2023). Meanwhile, mechanistic and integrative reviews have examined how airborne toxicants may contribute to the initiation or promotion of brain tumour development through mechanisms such as DNA damage, epigenetic alterations, microglial activation, and disruption of the BBB. These findings provide biological plausibility for the associations reported in epidemiological studies, but do not by themselves establish a causal relationship (Pagano et al., 2023; Ruszkiewicz et al., 2019).

Climate change is contributing to changes in wildfire seasons and may increase the duration and intensity of smoke exposure. A large Canadian cohort study reported an association between prolonged wildfire exposure and increased overall cancer incidence, alongside site-specific associations and null findings for haematological malignancies. These findings suggest that associations between wildfire exposure and cancer may differ across cancer types (Korsiak et al., 2022). Climate-related extreme events, including heatwaves, wildfires, and floods, may also disrupt neuro-oncology services, including diagnostic procedures, neurosurgical capacity, and radiotherapy scheduling. Such disruptions may delay diagnosis and compromise continuity of treatment, particularly in highly exposed or resource-constrained settings (Hassanipour et al., 2023; Korsiak et al., 2022; Pagano et al., 2023; Ruszkiewicz et al., 2019).

The implications for practice and policy include three main priorities. First, reducing emissions from combustion sources is essential to limit air pollution. Second, measures should be implemented to protect communities and workers who are particularly vulnerable to wildfire smoke and smog. Third, healthcare systems should develop and maintain preparedness plans to ensure continuity of neuro-oncology services during climate-related emergencies (Hassanipour et al., 2023; Korsiak et al., 2022; Pagano et al., 2023; Ruszkiewicz et al., 2019).

Prostate Cancer and Climate Change

Prostate cancer is the second most frequently diagnosed malignancy in men and a major cause of cancer-related mortality. Non-modifiable determinants, including age, ancestry, and family history, remain central considerations in this field, while evidence regarding modifiable factors, such as diet, physical activity, and environmental and occupational exposures, continues to evolve. In this context, climate-sensitive combustion-related pollutant mixtures, characterised by elevated concentrations of PM_{2.5} and NO₂, warrant consideration given their documented endocrine-disrupting activity and pro-inflammatory potential. As discussed by Youogo et al. (2022) and Zimta et al. (2021), various constituents, including polycyclic aromatic hydrocarbons (PAHs), benzene, and metals, may affect the prostate through several mechanisms involving oxidative stress, mitochondrial dysfunction, and immune signalling. These constituents may also disrupt systemic hormonal pathways. Endocrine-disrupting pollutants have been hypothesised to interact with metabolic syndrome, a condition whose prevalence may be influenced by climate-related changes in diet and lifestyle. This interaction could potentially increase susceptibility to prostate cancer (Youogo et al., 2022; Zimta et al., 2021).

Population-based data increasingly support this biological plausibility, although the strength and interpretation of the evidence require careful consideration. A Canadian population-based case-control study involving 1,420 cases and 1,424 controls observed an increased risk of prostate cancer associated with long-term exposure to PM_{2.5} and NO₂ (Youogo et al., 2022). In a large cohort of older adults in the United States, prolonged exposure to PM_{2.5} and NO₂ was associated with synergistic effects on the incidence of primary cancers, highlighting concerns about the combined impact of these pollutants, whose concentrations may be affected by climate-related changes (Wei et al., 2023). A case-control analysis conducted in Montreal using land-use regression also demonstrated an association between elevated NO₂ levels and an increased risk of prostate cancer (Parent et al., 2013). Considered within the broader context of industrialization, land use, and climate change, these findings support the view that environmental quality and cancer control are closely interconnected (Zimta et al., 2021).

Across all cancer types, several shared biological and systemic mechanisms emerge as central to the relationship between climate change and cancer. These include oxidative stress, chronic inflammation, endocrine disruption, immune dysregulation, and disruptions to healthcare delivery during extreme weather events. Integrating these shared pathways

provides a coherent framework for interpreting findings specific to individual cancer sites.

The implications of these findings include several priorities. Primary prevention requires reducing exposure to PM_{2.5} and NO₂ through reductions in emissions from traffic, industrial activities, and solid-fuel combustion (see Parent et al., 2013; Wei et al., 2023; Youogo et al., 2022; Zimta et al., 2021). Exposure mitigation is also particularly important; during episodes involving smog, dust, or wildfire smoke, alerts should be implemented, clean-air spaces should be provided, and appropriate respiratory protection should be made available to highly exposed groups and workers (Zimta et al., 2021). Maintaining continuity of clinical care is equally important. PSA testing, mpMRI and biopsy pathways, radiotherapy, and follow-up should be maintained during smoke and heat events through flexible scheduling and contingency plans (Zimta et al., 2021). Risk counselling should address the possibility that cumulative, long-term exposure to PM_{2.5} and NO₂ may modify prostate cancer risk, particularly among residents of highly exposed neighbourhoods, while acknowledging that stronger evidence is needed to establish causality (see Parent et al., 2013; Wei et al., 2023; Youogo et al., 2022). The following section will examine the relationship between surveillance and policy. To identify disparities and inform targeted emission-control policies, cancer registries should be linked with high-resolution air-quality data. Furthermore, research should prioritise low-dose exposure-response relationships, gene-environment interactions, windows of susceptibility, and personal and time-varying exposure measurements (Parent et al., 2013; Wei et al., 2023; Zimta et al., 2021).

Colorectal Cancer and Climate Change

Colorectal cancer (CRC) is the third most commonly diagnosed malignancy worldwide and a leading cause of cancer-related mortality. Established risk factors, including dietary patterns, obesity, physical inactivity, alcohol consumption, tobacco use, and inherited syndromes, remain central to prevention and control. However, a growing body of evidence suggests that climate-sensitive exposures may be relevant to CRC risk and outcomes through mechanisms involving inflammation, oxidative stress, microbiome perturbation, and metabolic dysregulation (Chu et al., 2021; Granados-Romero et al., 2017; Jenwitheesuk et al., 2020; Ku et al., 2021).

Air-pollution mixtures rich in PM_{2.5} and reactive gases are a major concern, as warming and drought can prolong wildfire seasons, intensify dust episodes, and favour secondary aerosol formation. A substantial body of research has reported a positive association between long-term exposure to PM_{2.5} and NO₂ and the incidence of colorectal cancer (CRC). This association has been observed in several large-scale population studies,

including satellite-based assessments in Thailand and multicentre cohorts that have also identified gene–environment interactions involving PM_{2.5} and CRC risk (Chu et al., 2021; Jenwitheesuk et al., 2020; Ku et al., 2021). Short-term increases in particulate pollution have also been associated with unfavourable outcomes among patients with CRC. This finding suggests that both chronic and acute exposure to particulate pollution may play a role in disease progression and CRC-related mortality (Ku et al., 2021).

Disruptions to food systems associated with climate change have the potential to affect food availability, safety, and affordability, potentially shifting dietary intake away from protective dietary patterns, such as those rich in fibre, towards greater consumption of processed foods. In addition, the levels of certain contaminants may increase under changing environmental conditions. Metabolomic profiling has shown that diet–environment interactions can modulate metabolic and signalling pathways relevant to colorectal cancer (CRC). Consequently, climate-driven changes in food quality, nutrient composition, and contaminant loads may alter the structure and function of the gut microbiome, potentially reducing beneficial short-chain fatty acids, disrupting bile-acid profiles, and promoting insulin resistance. These changes may, in turn, contribute to chronic inflammation and metabolic dysregulation, which intersect with established CRC-related pathways. Collectively, these mechanisms provide a biological rationale for the hypothesis that climate-related stressors may increase the population burden of CRC (Granados-Romero et al., 2017; Rattray et al., 2017).

The implications for CRC control include integrating cancer prevention and control strategies with measures to address air pollution. Primary prevention should involve the rapid reduction of emissions from combustion sources and the implementation of effective dust-control measures. Public guidance and protective measures should be provided for high-risk groups during smoke and smog events. During extreme weather events, plans should be implemented to maintain continuity of CRC screening, including flexible scheduling and remote pre-visit triage. In addition, nutrition policies that safeguard access to healthy, affordable foods should be implemented as climate pressures intensify (Chu et al., 2021; Granados-Romero et al., 2017; Jenwitheesuk et al., 2020; Ku et al., 2021; Rattray et al., 2017).

Liver Cancer and Climate Change

Primary liver cancer, predominantly represented by hepatocellular carcinoma (HCC), is a major contributor to global cancer mortality. Established risk factors for primary liver cancer include chronic hepatitis B and C infections, alcohol consumption, aflatoxin exposure, and the increasing burden of metabolic

dysfunction-associated steatotic liver disease (MASLD, formerly referred to as non-alcoholic fatty liver disease [NAFLD]). Climate-sensitive hazards intersect with these determinants through multiple pathways, including combustion-related air pollution, particularly PM_{2.5} and NO₂, heat and drought, which can affect food safety and increase the risk of fungal toxin contamination, and extreme weather events that disrupt prevention and healthcare delivery. Collectively, these factors may exacerbate systemic inflammation, oxidative stress, and lipid and metabolic dysregulation, all of which are implicated in the development of hepatocellular carcinoma (Gan et al., 2023; Saad-Hussein et al., 2022; So et al., 2021; VoPham, 2019).

The growing body of epidemiological evidence provides further support for these potential mechanisms. Multi-cohort analyses conducted in Europe have indicated an association between long-term exposure to ambient air pollution and an increased risk of several types of cancer, including liver cancer. These findings reinforce concerns about chronic exposure to fine particulate matter and co-pollutants (So et al., 2021). Several recent systematic reviews have reported positive associations between air pollution and liver cancer incidence and mortality. However, these reviews have also highlighted heterogeneity in exposure assessment and the control of confounding factors, underscoring the need for improved study designs and harmonized methods (Gan et al., 2023). Broader reviews examining climate change and liver disease have identified plausible links involving infectious, toxic, and metabolic pathways and have called for integrated surveillance systems, or health maps, that combine climate, air-quality, and liver-health indicators (Saad-Hussein et al., 2022; VoPham, 2019) (Gan et al., 2023b). Findings from complementary studies suggesting that large-scale climatic patterns may help explain geographical and temporal variations in the liver cancer burden have also prompted further research into the role of climate variability. These findings support the need for environmental epidemiological studies using higher-resolution data.

The implications of these findings include several priorities. For primary prevention, combustion emissions from traffic, industry, and solid-fuel use should be reduced to lower exposure to PM_{2.5} and NO₂, which have been associated with an increased risk of liver cancer. Food-safety systems should also be strengthened through improved drying and storage practices and enhanced surveillance to mitigate climate-sensitive aflatoxin contamination in staple crops (Gan et al., 2023a; Saad-Hussein et al., 2022; So et al., 2021; VoPham, 2019). Clinical and public health practices should anticipate and address disruptions to care during heat, smoke, or flood events. This includes maintaining hepatitis B virus (HBV) vaccination programmes,

screening high-risk populations, and ensuring continuity of antiviral therapy. Incorporating environmental exposure histories into liver clinic workflows is also recommended wherever feasible (Gan et al., 2023a; Saad-Hussein et al., 2022; VoPham, 2019). Research and surveillance should be strengthened by linking cancer registries with liver disease cohorts and integrating high-resolution air-quality and climate datasets. This approach requires prioritising improved exposure assessment, including personal and time-varying exposure metrics. Furthermore, gene–environment and metabolic interactions relevant to HCC associated with metabolic dysfunction-related liver disease should be investigated. These priorities are supported by the findings of Gan et al., 2023a, 2023b; Saad-Hussein et al., 2022; So et al., 2021; and VoPham, 2019.

Urological Cancers and Climate Change

Among the various urological malignancies, bladder cancer and kidney cancer show notable associations with climate-related environmental exposures. Climate change may exacerbate existing risks by contributing to the deterioration of air and water quality. Pollutants from traffic and industrial emissions, as well as wildfire smoke, may contribute to this deterioration. Additionally, contaminants such as arsenic and disinfection by-products in drinking water can adversely affect water quality and pose risks to human health. These environmental factors may compound established behavioural risk factors, particularly tobacco smoking (see Caballero et al., 2022; Dobruch & Oszczudłowski, 2021; Sakhvidi et al., 2020).

Bladder cancer is a major global health concern, and smoking is its primary modifiable risk factor. Ambient air pollution, which has been classified as carcinogenic to humans by the International Agency for Research on Cancer (IARC), may contribute to bladder carcinogenesis through exposure to polycyclic aromatic hydrocarbons and other combustion-related pollutant mixtures. Climate-related stressors that intensify droughts and floods may also compromise water systems, potentially increasing exposure to arsenic and disinfection by-products. Several population-based studies have reported positive associations between long-term exposure to hydrocarbons and PM_{2.5}, including certain metal components, and the incidence of bladder cancer. These findings are consistent with proposed biological mechanisms (see Caballero et al., 2022; Chen et al., 2022; Dobruch & Oszczudłowski, 2021; Sakhvidi et al., 2020; Zhang et al., 2022).

Renal cell carcinoma is the most common type of kidney cancer. The predominant histological subtype is clear cell renal cell carcinoma, although other subtypes also occur. Suspected environmental contributors include trichloroethylene, arsenic-contaminated water,

and certain pesticides, with additional evidence suggesting possible associations with traffic- and occupation-related pollutants, including petrol vapours, diesel exhaust, PAHs, and nitrogen oxides (NO_x). Experimental research indicates that ultrafine particles and diesel exhaust particles may reach the kidneys and induce biological processes relevant to carcinogenesis. These findings provide mechanistic support for epidemiological associations observed in several cohorts (Hvidtfeldt et al., 2022; Raaschou-Nielsen et al., 2017).

The implications for prevention and policy include reducing combustion emissions from traffic, industry, and solid-fuel use, as well as strengthening drinking-water systems. Monitoring and mitigating arsenic contamination and disinfection by-products are particularly important in regions affected by droughts and floods (see Caballero et al., 2022; Dobruch & Oszczudłowski, 2021; Sakhvidi et al., 2020). In clinical practice, brief environmental exposure histories should be incorporated into counselling for patients at risk or undergoing follow-up, including questions about occupational exposure to solvents such as trichloroethylene, long-term exposure to traffic and industrial pollution, and the use of private well water (Hvidtfeldt et al., 2022). Equity and preparedness should also be prioritised. During smoke and smog episodes, appropriate measures include issuing public alerts, establishing clean-air spaces, and promoting evidence-based respiratory protection. Maintaining continuity of diagnostic services and treatment during climate-related extreme events is equally important (see Caballero et al., 2022; Dobruch & Oszczudłowski, 2021; Sakhvidi et al., 2020). Research priorities include clarifying the dose–response relationship between PM_{2.5} exposure, its elemental composition, and bladder cancer; improving exposure assessment for nitrogen oxides (NO_x) and ultrafine particles in kidney cancer research; and evaluating climate-related extreme events, such as wildfire smoke episodes and water crises, as potential short-term peaks superimposed on chronic exposures (Chen et al., 2022; Hvidtfeldt et al., 2022; Raaschou-Nielsen et al., 2017; Zhang et al., 2022).

Cervical Cancer and Climate Change

Cervical cancer is one of the most common malignancies affecting women worldwide. As discussed by Scheurer et al. (2014), persistent infection with high-risk human papillomavirus (HPV) is the principal cause of cervical cancer. However, climate-sensitive exposures may influence disease risk and outcomes by affecting immune function, oxidative stress, endocrine signalling, and the continuity of healthcare services. In particular, combustion-related air pollution originating from traffic, industrial activities, diesel particulate matter (DPM), and

polycyclic aromatic hydrocarbons (PAHs) represents a growing concern in rapidly urbanizing regions and during wildfire or dust episodes (Scheurer et al., 2014). Numerous epidemiological studies have reported findings suggesting an association between ambient air pollutants and cervical cancer. Research has indicated that elevated levels of NO_x and NO₂, together with the presence of benzene, PAHs, and DPM in residential environments, may be associated with an increased risk of cervical cancer. This association has also been investigated in studies examining exposure to diesel exhaust and the subsequent development of cervical cancer in cohorts of women (Scheurer et al., 2014). A recent cross-national analysis suggests that the economic context may modify these associations. The study found stronger associations between population-level air pollution and cervical and breast cancer incidence in countries with lower gross domestic product (GDP). This finding suggests that these countries may be more vulnerable when resources for screening and treatment are limited (Hu et al., 2023).

Beyond potential direct carcinogenic effects and immune modulation, climate-related extreme events, including periods of intense wildfire smoke, heatwaves, and floods, may disrupt HPV vaccination, screening through Pap tests or HPV testing, diagnostic follow-up, and timely treatment. Such disruptions may contribute to later-stage diagnosis and poorer outcomes, particularly in settings where healthcare resources are already limited (Hu et al., 2023; Scheurer et al., 2014).

The implications for prevention and healthcare delivery include several priorities. Primary prevention should involve reducing exposure to NO_x and particulate matter through reductions in traffic and industrial emissions and solid-fuel combustion. Public guidance should also be issued during smoke and smog events to help protect exposed populations (Hu et al., 2023; Scheurer et al., 2014). Maintaining continuity of screening and vaccination is particularly important in this context. During climate-related emergencies, HPV vaccination, screening, and treatment services should be maintained through mobile outreach strategies, flexible scheduling, backup power supplies, and air filtration systems in clinics (Hu et al., 2023). Equity should also be prioritized by focusing on communities with high pollution exposure and limited resources, where environmental burdens may coincide with disruptions to healthcare services. Brief environmental exposure histories should also be incorporated into patient counselling where appropriate (Hu et al., 2023; Scheurer et al., 2014). Research and surveillance should be strengthened to clarify exposure–response relationships and investigate how socioeconomic context influences outcomes. This requires linking cancer registries with high-resolution air-quality

datasets and records of extreme weather events (Arnaoutoglou et al., 2023; Hu et al., 2023).

Ovarian Cancer and Climate Change

Ovarian cancer is one of the most lethal gynaecological malignancies. Non-modifiable risk factors, including age, BRCA1/2 mutations, and family history, as well as reproductive factors, such as parity, oral contraceptive use, and breastfeeding, remain central to understanding ovarian cancer risk. Meanwhile, climate-sensitive exposures, particularly combustion-derived air pollution, including PM_{2.5}, NO₂, and polycyclic aromatic hydrocarbons (PAHs), as well as smoke events, may influence disease risk and outcomes through mechanisms involving oxidative stress, endocrine disruption, and immune modulation (Arnaoutoglou et al., 2023).

A growing body of population-based research has reported positive associations between long-term exposure to PM_{2.5} and NO₂ and ovarian cancer incidence or mortality. However, the magnitude of these associations varies, and some estimates include the null value, potentially reflecting exposure misclassification and differences in confounding control across cohorts (Arnaoutoglou et al., 2023; Dehghani et al., 2022; Yu et al., 2021). Pollutant mixtures containing PAHs, benzene, and metals originating from traffic, industrial activities, and wildfires may also be biologically relevant, given established links between these pollutants and inflammation, DNA damage, and altered steroidogenesis in ovarian tissue (Arnaoutoglou et al., 2023; Dehghani et al., 2022; Yu et al., 2021).

The consequences of disruptions to healthcare delivery and the resulting vulnerabilities are significant. Climate-related extreme events, including heatwaves, wildfire smoke episodes, and floods, may adversely affect gynaecological oncology care pathways, including diagnostic imaging, the scheduling of cytoreductive surgery, chemotherapy administration, and follow-up (see Badwelan et al., 2023; Yu et al., 2021). The effects of such events may be disproportionately experienced in resource-constrained settings and among patients with limited access to specialist care.

The implications for prevention, clinical practice, and research include several priorities. Primary prevention should focus on accelerating reductions in traffic, industrial, and solid-fuel emissions to lower concentrations of PM_{2.5} and NO₂. Public guidance should also be disseminated during periods of smoke and smog to help protect high-risk groups (Arnaoutoglou et al., 2023; Badwelan et al., 2023; Dehghani et al., 2022; Yu et al., 2021). Maintaining continuity of clinical care is particularly important in this context. During extreme events, contingency plans should be developed to incorporate flexible scheduling,

backup power supplies, air filtration, and remote pre-assessment. These measures are important for maintaining diagnostic procedures, surgical services, and chemotherapy during emergencies (Badwelan et al., 2023; Yu et al., 2021). Risk counselling and equity should also be prioritized. In the absence of conclusive evidence, cumulative exposure to air pollution may be considered a potential risk modifier in highly exposed neighbourhoods. Furthermore, support should be prioritized for patients facing geographical or socioeconomic barriers to timely gynaecological oncology care (Arnaoutoglou et al., 2023; Badwelan et al., 2023; Dehghani et al., 2022; Yu et al., 2021). Research and surveillance should be strengthened by integrating ovarian cancer registries with high-resolution air-quality and extreme-event datasets. This integration would facilitate improved assessment of personal and time-varying exposures and support the evaluation of pollutant-mixture effects and gene-environment interactions (Arnaoutoglou et al., 2023; Badwelan et al., 2023; Dehghani et al., 2022; Yu et al., 2021).

Oral Cancer and Climate Change

Oral cavity squamous cell carcinoma (OCSCC) remains a major global health burden. Established risk factors include tobacco use, alcohol consumption, and betel quid or areca nut use, with human papillomavirus (HPV) contributing to the development of cancers at selected subsites. In addition to these established risks, climate-sensitive exposures, particularly combustion-derived air pollution, including PM_{2.5}, NO₂, and polycyclic aromatic hydrocarbons (PAHs), wildfire smoke, dust storms, and ultraviolet (UV) radiation in relation to lip cancer, may influence disease incidence and outcomes through mechanisms involving oxidative stress, immune modulation, endocrine disruption, and disruptions to healthcare delivery (Khanna, 2010; Patil, 2023; Yin et al., 2020).

Population-based studies increasingly report positive associations between long-term exposure to PM_{2.5} and NO₂ and the incidence or mortality of oral and oropharyngeal cancers, although the magnitude of these associations varies across cohorts and exposure-assessment models (Khanna, 2010; Patil, 2023). Associations have also been reported in settings where pollutant mixtures contain elevated levels of PAHs, benzene, and metals from traffic, industrial activities, or biomass burning. The composition and concentration of these pollutant mixtures may be affected by warming and changes in wildfire frequency and intensity (Khanna, 2010; Patil, 2023; Yin et al., 2020). For lip cancer, chronic UV exposure remains an important environmental hazard, particularly among outdoor workers and populations living in regions with high levels of solar radiation (Sadeghi et al., 2023). Limited evidence also suggests that household solid-fuel smoke

and episodic wildfire smoke may increase short-term symptom burden and potentially contribute to disease progression through mucosal inflammation, although causal relationships remain uncertain (Khanna, 2010; Patil, 2023; Yin et al., 2020).

Climate-related extreme events, including heatwaves, smoke episodes, and floods, may interrupt screening, biopsy procedures, imaging, surgery, radiotherapy, and rehabilitation, potentially prolonging the time to diagnosis and treatment. Such disruptions may exacerbate existing disparities, particularly in communities where access to dental services and specialist head-and-neck oncology care is already limited (Nagel et al., 2018; Sadeghi et al., 2023; Yin et al., 2020).

The implications for prevention, clinical practice, equity, and surveillance include several priorities. Primary prevention should focus on reducing emissions from traffic, industrial activities, and solid-fuel combustion to lower exposure to PM_{2.5}, NO₂, and PAHs. Occupational UV protection should also be strengthened through measures such as providing shade structures and appropriate lip and skin protection for outdoor workers (Khanna, 2010; Patil, 2023; Sadeghi et al., 2023; Yin et al., 2020). Maintaining continuity of clinical care during climate-related emergencies is equally important. Diagnostic and treatment services should be maintained through flexible scheduling, remote pre-assessment, backup power supplies, air filtration in clinics, and prioritized care pathways for urgent head-and-neck cases (Nagel et al., 2018; Sadeghi et al., 2023; Yin et al., 2020). Risk counselling should address cumulative exposure to air pollution and UV radiation as potential risk modifiers, particularly among residents of highly exposed neighbourhoods and people working outdoors, while acknowledging that stronger causal evidence is needed (Khanna, 2010; Patil, 2023; Sadeghi et al., 2023; Yin et al., 2020). Equity and surveillance should also be prioritized by linking cancer registries with high-resolution air-quality, UV, and extreme-event datasets to identify areas of elevated exposure and vulnerability. Brief environmental exposure histories should also be incorporated into routine oral oncology assessments (Nagel et al., 2018; Sadeghi et al., 2023; Yin et al., 2020). Research priorities include improving the assessment of personal and time-varying exposures, evaluating the combined effects of pollutant mixtures involving PAHs, metals, and ozone, and investigating the acute effects of wildfire smoke superimposed on chronic exposure and their potential influence on disease progression (Khanna, 2010; Nagel et al., 2018; Patil, 2023; Sadeghi et al., 2023; Yin et al., 2020).

Gastric Cancer and Climate Change

Gastric cancer remains a leading cause of cancer-related mortality worldwide. Established risk factors

include *Helicobacter pylori* (*H. pylori*) infection, diets high in salted or preserved foods, smoking, certain genetic factors, and socioeconomic circumstances. In addition to these established determinants, climate-sensitive exposures, particularly combustion-derived air pollution, including PM_{2.5} and NO₂, and water-quality stressors, such as nitrates and nitrites that may contribute to endogenous nitrosation, could influence disease risk and outcomes through oxidative stress, chronic inflammation, disruption of the gut microbiome, and interruptions to healthcare delivery (Dehghani et al., 2021; He et al., 2022; Lehrer & Rosenzweig, 2014).

Population-based studies increasingly report positive associations between long-term exposure to PM_{2.5} and NO₂ and gastric cancer incidence or mortality, although the magnitude of these associations varies across studies, partly because of differences in exposure assessment and the control of confounding factors (Dehghani et al., 2021; Lehrer & Rosenzweig, 2014). Climate change may intensify runoff and flooding, potentially increasing nitrate levels in surface water and groundwater. Higher nitrate intake has been associated with the formation of endogenous N-nitroso compounds and an increased risk of gastric cancer in several settings (He et al., 2022; Maleki et al., 2023). These epidemiological findings are consistent with proposed mechanisms involving inflammatory and nitrosative pathways, interactions with *H. pylori*-associated gastritis, and exposure to pollutant mixtures containing polycyclic aromatic hydrocarbons (PAHs), benzene, and metals from traffic and industrial sources (Dehghani et al., 2021; He et al., 2022; Lehrer & Rosenzweig, 2014; Maleki et al., 2023).

Climate-related extreme events, including heatwaves, wildfire smoke episodes, and floods, may interrupt *H. pylori* testing and eradication, delay endoscopy and pathological assessment, and disrupt perioperative care and chemotherapy services, particularly in resource-constrained regions. Such disruptions may contribute to later-stage diagnosis and poorer outcomes, especially in populations where access to healthcare is already limited (He et al., 2022; Maleki et al., 2023; Naqvi et al., 2023).

The implications for prevention, clinical practice, risk counselling, and health equity include several priorities. Primary prevention should include reducing emissions from traffic, industrial activities, and solid-fuel combustion to lower exposure to PM_{2.5} and NO₂, alongside strengthening water-quality management through the monitoring and mitigation of nitrate and nitrite contamination as climate-related hydrological extremes intensify (Dehghani et al., 2021; He et al., 2022; Lehrer & Rosenzweig, 2014; Maleki et al., 2023). Continuity of clinical care should be maintained during smoke, heat, and flood events by developing

contingency plans for *H. pylori* testing and treatment, endoscopic screening and diagnostic services, and adjuvant therapy. Measures may include flexible scheduling, backup power supplies, air filtration, and mobile outreach services (He et al., 2022; Maleki et al., 2023; Naqvi et al., 2023). Risk counselling should acknowledge cumulative exposure to air pollution and nitrates as potential risk modifiers, particularly among residents of highly exposed neighbourhoods and populations in agricultural regions, while recognizing that stronger causal evidence is needed (Dehghani et al., 2021; He et al., 2022; Lehrer & Rosenzweig, 2014; Maleki et al., 2023). Research and surveillance should prioritise linking gastric cancer registries with high-resolution air-quality, hydrological, and extreme-event datasets, improving the assessment of personal and time-varying exposures, and evaluating the combined effects of pollutant mixtures and potential interactions between *H. pylori* infection and pollution exposure (Dehghani et al., 2021; He et al., 2022; Lehrer & Rosenzweig, 2014; Maleki et al., 2023; Naqvi et al., 2023).

Thyroid Cancer and Climate Change

Thyroid cancer, predominantly papillary thyroid carcinoma, has shown increasing incidence in many regions. Established determinants include exposure to ionising radiation, particularly during childhood, female sex, and genetic susceptibility. Although overdiagnosis accounts for part of the increase in reported incidence, it does not fully explain recent trends. In addition to these established factors, climate-sensitive exposures may influence disease risk and outcomes through endocrine disruption, oxidative stress, immune modulation, and disruptions to healthcare delivery (Parker, 2021; Puri et al., 2017).

Population-based studies increasingly report positive associations between long-term exposure to PM_{2.5} and NO₂ and thyroid cancer incidence or mortality, although the magnitude of these associations varies across cohorts and exposure-assessment models (Parker, 2021; Puri et al., 2017; Wright et al., 2019). Hydrological changes that increase nitrate concentrations in drinking water are of potential concern because nitrate can compete with iodide for transport by the sodium-iodide symporter. This may affect thyroid hormone synthesis and potentially increase thyroid-stimulating hormone (TSH) levels, thereby stimulating proliferative signalling in the thyroid. Several ecological and cohort studies have reported associations between higher nitrate intake and increased thyroid cancer risk (Hill et al., 2023; Wright et al., 2019). Broader reviews of endocrine-disrupting chemicals (EDCs), some of which may be redistributed or mobilized by warming and extreme weather events, also highlight potential disruptions to thyroid hormone pathways relevant to tumorigenesis. However, the

causal relationships between these exposures and thyroid cancer remain uncertain (Hill et al., 2023; Parker, 2021; Wright et al., 2019).

Climate-related events, including smoke episodes, dust storms, heatwaves, and floods, may disrupt the evaluation of thyroid nodules, access to ultrasound and fine-needle aspiration (FNA), surgical scheduling, radioiodine supply chains, and follow-up care. These disruptions may delay definitive treatment, particularly in resource-constrained settings where healthcare services and supply chains are already vulnerable (Parker, 2021; Schwerdtle et al., 2018).

The implications for prevention, clinical practice, risk counselling, and health equity include several priorities. Primary prevention should include reducing emissions from traffic, industrial activities, and solid-fuel combustion to lower exposure to PM_{2.5} and NO₂, alongside strengthening water-quality monitoring and implementing measures to mitigate nitrate contamination in vulnerable watersheds (Hill et al., 2023; Parker, 2021; Puri et al., 2017; Wright et al., 2019). Continuity of clinical care should be maintained during climate-related emergencies by protecting access to ultrasound, FNA, pathology services, surgery, and radioiodine through flexible scheduling, backup power supplies, air filtration, and resilient supply chains (Parker, 2021; Schwerdtle et al., 2018). Risk counselling should consider cumulative exposure to air pollution and nitrates as potential risk modifiers, particularly among residents of highly exposed neighbourhoods and populations in agricultural regions, while acknowledging that stronger causal evidence is needed (Hill et al., 2023; Parker, 2021; Puri et al., 2017; Wright et al., 2019). Research and surveillance should prioritize linking thyroid cancer registries with high-resolution air-quality, hydrological, and extreme-event datasets, improving the assessment of personal and time-varying exposures, and evaluating the combined effects of exposure mixtures and potential interactions with iodine status (Hill et al., 2023; Parker, 2021; Schwerdtle et al., 2018; Wright et al., 2019).

Skin Cancer and Climate Change

Skin cancers, including keratinocyte carcinomas (basal cell carcinoma and squamous cell carcinoma) and melanoma, are among the malignancies most directly influenced by climate-sensitive environmental exposures because of their established relationship with ultraviolet (UV) radiation. Climate change may modify ambient UV exposure through meteorological conditions and stratospheric ozone dynamics, alter behavioural patterns by increasing outdoor activity during warmer seasons, and affect co-exposures such as air pollutants and wildfire smoke. These factors may influence cutaneous inflammation, oxidative stress, and immune function (PRESS RELEASE N° 221 IARC: Outdoor Air Pollution a Leading Environmental Cause

of Cancer Deaths, 2013; Silva, 2025; Turner et al., 2020).

Decades of epidemiological research have demonstrated strong associations between intermittent, high-intensity UV exposure and melanoma, and between cumulative UV exposure and squamous cell carcinoma. Basal cell carcinoma has been associated with patterns of both intermittent and cumulative exposure (PRESS RELEASE N° 221 IARC: Outdoor Air Pollution a Leading Environmental Cause of Cancer Deaths, 2013; Silva, 2025). Projected warming and changes in cloud cover in some regions, together with shifts in holiday-related and occupational outdoor activities, may increase cumulative lifetime UV exposure and the frequency of sunburn, particularly during childhood and adolescence (PRESS RELEASE N° 221 IARC: Outdoor Air Pollution a Leading Environmental Cause of Cancer Deaths, 2013; Silva, 2025; Turner et al., 2020). Air-pollution mixtures containing polycyclic aromatic hydrocarbons (PAHs), nitrogen oxides, and particulate matter may create photosensitising and pro-oxidant conditions in the skin, potentially enhancing UV-mediated damage and affecting photoaging pathways implicated in carcinogenesis. Wildfire smoke introduces complex mixtures of organic aerosols that may contribute to similar biological processes (Mufson, n.d.; Turner et al., 2020). Although stratospheric ozone is recovering globally, regional and seasonal variability persists, and interactions between climate and ozone dynamics may alter surface UV levels in specific locations. These variations highlight the need for local monitoring rather than assumptions based on global trends (PRESS RELEASE N° 221 IARC: Outdoor Air Pollution a Leading Environmental Cause of Cancer Deaths, 2013; Silva, 2025; Turner et al., 2020).

Climate-related extreme events, including heatwaves, smoke episodes, and floods, may disrupt dermatology services, access to biopsies, and the scheduling of surgery and radiotherapy, potentially delaying diagnosis and treatment. These disruptions may disproportionately affect regions with limited dermatological capacity and people working in outdoor occupations (Mufson, n.d.; US EPA, n.d.).

The implications for prevention, clinical practice, occupational protection, and research include several priorities. Primary prevention should strengthen UV-protection measures, including the provision of shade in schools and workplaces, sun-safe scheduling of outdoor activities, protective clothing, and guidance on the use of broad-spectrum sunscreen. Public UV Index alerts should also be maintained and adapted to local conditions (PRESS RELEASE N° 221 IARC: Outdoor Air Pollution a Leading Environmental Cause of Cancer Deaths, 2013; Silva, 2025; Turner et al., 2020). Reducing emissions from combustion sources may

lower exposure to PAHs and particulate matter that could contribute to UV-related skin damage, while clear public guidance should be provided during wildfire-smoke and air-pollution episodes (Mufson, n.d.; Turner et al., 2020). Continuity of clinical care should be supported through contingency plans for biopsies, excisions, and radiotherapy during heatwaves and smoke events, including flexible scheduling, backup power supplies, air filtration, and remote triage of suspicious lesions (Mufson, n.d.; US EPA, n.d.). Particular attention should be given to outdoor workers and communities experiencing high levels of UV radiation or air pollution to ensure equitable access to preventive measures and diagnostic services (Mufson, n.d.; PRESS RELEASE N° 221 IARC: Outdoor Air Pollution a Leading Environmental Cause of Cancer Deaths, 2013; Silva, 2025; Turner et al., 2020; US EPA, n.d.). Research and surveillance should prioritize linking cancer registries with high-resolution UV, air-quality, and extreme-event datasets, improving the assessment of personal and time-varying exposures, and investigating the combined effects of UV radiation and air pollution (Mufson, n.d.; Turner et al., 2020; US EPA, n.d.). Although this review highlights growing evidence of links between climate-related factors and cancer, several methodological limitations in the existing literature warrant attention. Many studies rely on ecological designs that may be affected by exposure misclassification, inadequate control of confounding factors, and inconsistencies in outcome ascertainment. Differences in study design and pollutant measurement also limit the interpretation of meta-analyses. Future research should adopt harmonized exposure metrics, longitudinal study designs, and integrated environmental and health surveillance systems to strengthen causal inference.

Conclusion

Climate change is reshaping when, where, and how people are exposed to carcinogenic and co-carcinogenic agents. The most consistent patterns involve combustion-related air pollution across multiple cancer sites, ultraviolet radiation in relation to skin cancers, and climate-related disruptions to screening and treatment that may worsen outcomes. Although effect estimates vary according to population characteristics, exposure-assessment methods, and the control of confounding factors, converging epidemiological findings and established biological mechanisms, including oxidative stress, inflammation, immune and endocrine disruption, and changes in the microbiome, support the integration of climate and environmental considerations into cancer control strategies. This requires parallel efforts to reduce harmful exposures, strengthen the resilience of healthcare delivery, and address health inequities, as climate hazards and gaps in healthcare provision often affect the same communities.

Climate change should therefore be understood not as an isolated determinant of cancer risk, but as a risk multiplier that may exacerbate existing disparities in exposure, prevention, and treatment. Integrating climate resilience into cancer control policies is consequently essential to reducing future inequities in the global cancer burden.

Recommendations

Policy and population health measures should prioritize reducing emissions from traffic, industrial activities, and solid-fuel combustion to decrease exposure to particulate matter and nitrogen oxides. Drinking-water safety should be protected during droughts and floods, with particular attention to arsenic and nitrate/nitrite contamination, while food-safety systems should be strengthened to limit contamination by harmful substances. UV-protective environments should also be promoted through the provision of shade, appropriate work–rest schedules, protective clothing, and locally calibrated UV alerts. These recommendations are consistent with the World Health Organization’s Climate Change and Health Action Plan (2023) and align with United Nations Sustainable Development Goal 13 on Climate Action. Integrating these frameworks into cancer prevention and control can help align public health strategies with wider commitments to climate adaptation and health-system resilience.

Health-system preparedness and clinical practice should focus on maintaining continuity of imaging, endoscopy, radiotherapy, infusion services, surgery, and pathology during smoke episodes, heatwaves, and floods. Measures should include flexible scheduling, backup power supplies, air filtration, and clear contingency protocols. Brief environmental exposure histories should also be incorporated into oncology and high-risk clinical assessments to inform risk counselling and follow-up. Prevention programmes should be maintained during climate-related emergencies, including human papillomavirus (HPV) vaccination and screen-and-treat programmes, *Helicobacter pylori* (*H. pylori*) testing and treatment, and hepatitis B virus (HBV) vaccination and continuity of antiviral therapy. Adaptive interventions may include installing backup power and air-filtration systems in radiotherapy and infusion centres, safeguarding oncology medicine supply chains, and developing climate-resilient hospitals capable of maintaining essential diagnostic and treatment services during heatwaves, floods, and wildfire events.

Equity-focused interventions should prioritize highly exposed, resource-constrained communities by providing timely alerts, clean-air shelters, mobile outreach services, and transport assistance. Implementation of climate and health interventions

should also include monitoring and reporting equity indicators related to access to care, timeliness of diagnosis and treatment, and health outcomes to identify and address disparities.

Research, data collection, and surveillance should prioritize linking cancer registries with high-resolution environmental datasets covering air quality, UV radiation, hydrological conditions, and extreme weather events to characterize spatial patterns of risk and dose–response relationships, including at relatively low exposure levels. Exposure science should be advanced through personal and time-varying exposure measurements, mixture analyses, and investigations of gene–environment interactions and life-course windows of susceptibility. Real-world interventions, including clinical air filtration, smoke and UV alerts, and water remediation, should be evaluated using implementation-science frameworks, with findings systematically informing policy development and clinical practice.

Declarations

Ethics Approval and Consent to Participate

Ethics approval was not required for this review because it was based exclusively on previously published studies and did not involve human participants, the collection of new data from individuals, or access to identifiable personal information. Therefore, informed consent to participate was not applicable.

Consent for Publication

Not applicable.

Availability of Data and Material

No new data was generated or analyzed in this study.

Conflicts of Interest / Competing Interests

The authors declare that there are no conflicts of interest.

Funding

The authors declare that this research received no external funding.

Authors' Contributions

A.F.A.: Conceptualization, Methodology, Writing – original draft, Project administration.

S.H.A.: Conceptualization, Methodology, Writing – original draft, Project administration.

S.N.: Methodology, Formal analysis, Investigation, Data curation, Writing – review & editing.

S.A.: Software, Formal analysis, Investigation, Writing – review & editing.

A.H.: Formal analysis, Investigation, Resources, Funding acquisition.

M.A.E.: Formal analysis, Data curation, Visualization, Funding acquisition.

S.E.B.I.: Investigation, Resources, Data curation, Funding acquisition.

M.H.N.: Investigation, Resources, Data curation, Funding acquisition.

S.M.J.: Validation, Writing – review & editing, Visualization.

Acknowledgment

Not applicable

Use of Generative AI and AI-Assisted Technologies

Not applicable

References

- Arnaoutoglou, C., Dampala, K., Anthoulakis, C., Papanikolaou, E. G., Tentas, I., Dragoutsos, G., Machairiotis, N., Zarogoulidis, P., Ioannidis, A., Matthaios, D., Perdikouri, E. I., Giannakidis, D., Sardeli, C., Petousis, S., Oikonomou, P., Nikolaou, C., Charalampidis, C., & Sapalidis, K. (2023). Epithelial Ovarian Cancer: A Five Year Review. *Medicina*, 59, 1–12. <https://doi.org/10.3390/medicina59071183>
- Badwelan, M., Muaddi, H., Ahmed, A., Lee, K. T., & Tran, S. D. (2023). Oral Squamous Cell Carcinoma and Concomitant Primary Tumors, What Do We Know? A Review of the Literature. *Curr. Oncol.*, 30, 3721–3734. <https://doi.org/10.3390/currenco130040283>
- Caballero, J. M., Perez-Marquez, M., Gili, J. M., Pereira, J. C., Gomariz, A., Castillo, C., & Martin-Baranera, M. (2022). Environmental Factors Involved in the High Incidence of Bladder Cancer in an Industrialized Area in North-Eastern Spain. *Journal of Environmental and Public Health*, 2022, 1–11. <https://doi.org/10.1155/2022/1051046>
- Chen, J., Rodopoulou, S., Strak, M., Hoogh, K. de, Taj, T., Poulsen, A. H., Andersen, Z. J., Bellander, T., Brandt, J., Zitt, E., Fecht, D., Forastiere, F., Gulliver, J., Hertel, O., Hoffmann, B., Hvidtfeldt, U. A., Verschuren, W. M. M., Jorgensen, J. T., Katsouyanni, K., ... Hoek, G. (2022). Long-term exposure to ambient air pollution and bladder cancer incidence in a pooled European cohort: the ELAPSE project. *Br J Cancer*, 126(10), 1499–1507. <https://doi.org/10.1038/s41416-022-01735-4>
- Chu, H., Xin, J., Yuan, Q., Wu, Y., Du, M., Zheng, R., Liu, H., Wu, S., Zhang, Z., & Wang, M. (2021). A prospective study of the associations among fine particulate matter, genetic variants, and the risk of

colorectal cancer. *Environment International*, 147, 1–8. <https://doi.org/10.1016/j.envint.2020.106309>

Cruz, R. S. da, Carney, E. J., Clarke, J., Cao, H., Cruz, M. I., Benitez, C., Jin, L., Fu, Y., Cheng, Z., Wang, Y., & Assis, S. de. (2018). Paternal malnutrition programs breast cancer risk and tumor metabolism in offspring. *Breast Cancer Res*, 20(1), 99. <https://doi.org/10.1186/s13058-018-1034-7>

Dehghani, S., Abedinzade, A., & Vali, M. (2021). Ambient air pollution exposure and thyroid cancer incidence in Iran. *Journal of Air Pollution and Health*, 6(1), 30–41. <https://doi.org/10.18502/japh.v6i1.7603>

Dehghani, S., Moshfeghinia, R., Ramezani, M., Vali, M., Oskoei, V., Amiri-Ardekani, E., & Hopke, P. (2022). Exposure to air pollution and risk of ovarian cancer: a review. *Rev Environ Health*, 1–12. <https://doi.org/10.1515/reveh-2021-0129>

Dobrush, J., & Oszczudłowski, M. (2021). Bladder Cancer: Current Challenges and Future Directions. *Medicina*, 57(8), 749. <https://doi.org/10.3390/medicina57080749>

Ennour-Idrissi, K., Ayotte, P., & Diorio, C. (2019). Persistent Organic Pollutants and Breast Cancer: A Systematic Review and Critical Appraisal of the Literature. *Cancers (Basel)*, 11(8), 1063. <https://doi.org/10.3390/cancers11081063>

Fernandez, E. (n.d.). *Climate Change Will Give Rise to More Cancers* | UC San Francisco. Retrieved <https://www.ucsf.edu/news/2020/11/418976/climate-change-will-give-rise-more-cancers>

Gan, T., Bambrick, H., Tong, S., & Hu, W. (2023a). Air pollution and liver cancer: A systematic review. *J Environ Sci (China)*, 126, 817–826. <https://doi.org/10.1016/j.jes.2022.05.037>

Gan, T., Bambrick, H., Tong, S., & Hu, W. (2023b). Does global warming increase the risk of liver cancer in Australia? Perspectives based on spatial variability. *Science of The Total Environment*, 859, 160412. <https://doi.org/10.1016/j.scitotenv.2022.160412>

Granados-Romero, J. J., Valderrama-Trevino, A. I., Contreras-Flores, E. H., Barrera-Mera, B., Enriquez, M. H., Uriarte-Ruiz, K., Ceballos-Villalva, J. C., Estrada-Mata, A. G., Rodriguez, C. A., & Arauz-Pena, G. (2017). Colorectal cancer: a review. *International Journal of Research in Medical Sciences*, 5(11), 4667–4676. <https://doi.org/10.18203/2320-6012.ijrms20174914>

Guo, T., Chen, S., Wang, Y., Zhang, Y., Du, Z., Wu, W., Chen, S., Ju, X., Li, Z., Jing, Q., Hao, Y., & Zhang, W. (2024). Potential causal links of long-term air pollution with lung cancer incidence: From the perspectives of mortality and hospital admission in a large cohort study in southern China. *Int J Cancer*, 154(2), 251–260. <https://doi.org/10.1002/ijc.34699>

Hassanipour, S., Nikbakht, H.-A., Amrane, A., Arab-Zozani, M., Shojaie, L., Rostami, S., & Badeenezhad, A. (2023). The Relationship between Air

Pollution and Brain Cancer: A Systematic Review and Meta-Analysis. *Annals of Global Health*, 89(1), 45, 1–13. <https://doi.org/10.5334/aogh.3889>

He, Q., Wu, M., Shi, Q., Tan, H., Wei, B., Tang, N., Chen, J., Liu, M., Duan, S., Chang, S., & Huang, P. (2022). Association of Ozone Exposures with the risk of thyroid nodules in Hunan Province: a population-based cohort study. *Environ Health*, 21(1), 65. <https://doi.org/10.1186/s12940-022-00874-8>

Hiatt, R. A., & Beyeler, N. (2023). Women's cancers and climate change. *International Federation of Gynecology and Obstetrics*, 160, 374–377. <https://doi.org/10.1002/ijgo.14407>

Hill, W., Lim, E. L., & Weeden, C. E. (2023). Lung adenocarcinoma promotion by air pollutants. *Nature*, 616, 159–167. <https://doi.org/10.1038/s41586-023-05874-3>

Hinchliffe, A., Kogevinas, M., Perez-Gomez, B., Ardanaz, E., Amiano, P., Marcos-Delgado, A., Castano-Vinyals, G., Llorca, J., Moreno, V., Alguacil, J., Fernandez-Tardon, G., Salas, D., Marcos-Gragera, R., Aragones, N., Guevara, M., Gil, L., Martin, V., Benavente, Y., Gomez-Acebo, I., ... Turner, M. C. (2021). Occupational Heat Exposure and Breast Cancer Risk in the MCC-Spain Study. *Cancer Epidemiol Biomarkers Prev*, 30(2), 364–372. <https://doi.org/10.1158/1055-9965.EPI-20-0732>

Hu, M., Jiang, C., Meng, R., Luo, Y., Wang, Y., Huang, M., Li, F., & Ma, H. (2023). Effect of air pollution on the prevalence of breast and cervical cancer in China: a panel data regression analysis. *Environmental Science and Pollution Research*, 30(34), 82031–82044. <https://doi.org/10.1007/s11356-023-28068-w>

Hvidtfeldt, U. A., Taj, T., Chen, J., Rodopoulou, S., Strak, M., Hoogh, K. de, Andersen, Z. J., Bellander, T., Brandt, J., Fecht, D., Forastiere, F., Gulliver, J., Hertel, O., Hoffmann, B., Jorgensen, J. T., Katsouyanni, K., Ketzel, M., Lager, A., Leander, K., ... Raaschou-Nielsen, O. (2022). Long term exposure to air pollution and kidney parenchyma cancer - Effects of low-level air pollution: a Study in Europe (ELAPSE). *Environmental Research*, 215, 1–10. <https://doi.org/10.1016/j.envres.2022.114385>

Jenwitheesuk, K., Peansukwech, U., & Jenwitheesuk, K. (2020). Accumulated ambient air pollution and colon cancer incidence in Thailand. *Scientific Reports*, 10, 1–12. <https://doi.org/10.1038/s41598-020-74669-7>

Khanna, S. (2010). Climate Change & Oral Health: Current Challenges & Future Scope. *International Journal of Environmental Science and Development*, 1(2), 190–192. <https://doi.org/10.7763/IJESD.2010.V1.35>

Korsiak, J., Pinault, L., Christidis, T., Burnett, R. T., Abrahamowicz, M., & Weichenthal, S. (2022). Long-term exposure to wildfires and cancer incidence

in Canada: a population-based observational cohort study. *Lancet Planet Health*, 6, 400–409. [https://doi.org/10.1016/S2542-5196\(22\)00067-5](https://doi.org/10.1016/S2542-5196(22)00067-5)

Ku, M.-S., Liu, C.-Y., Hsu, C.-Y., Chiu, H.-M., Chen, H.-H., & Chan, C.-C. (2021). Association of Ambient Fine Particulate Matter (PM_{2.5}) with Elevated Fecal Hemoglobin Concentration and Colorectal Carcinogenesis: A Population-Based Retrospective Cohort Study. *Cancer Control*, 28, 1–10. <https://doi.org/10.1177/10732748211041232>

Lehrer, S., & Rosenzweig, K. E. (2014). Cold Climate Is a Risk Factor for Thyroid Cancer. *Clin Thyroidol*, 26, 273–276. <https://doi.org/10.1089/ct.2014.26.273-276>

Ma, C., Jung, C.-R., Nakayama, S. F., Tabuchi, T., Nishihama, Y., Kudo, H., Morishima, T., Ohno, Y., Sobue, T., & Miyashiro, I. (2023). Short-term association of air pollution with lung cancer mortality in Osaka, Japan. *Environmental Research*, 224, 115503. <https://doi.org/10.1016/j.envres.2023.115503>

Maleki, Z., Hassanzadeh, J., Mendez-Arriaga, F., & Ghaem, H. (2023). Environmental factors and incidence of thyroid cancer in the world (1990-2019): an ecological study. *Environ Sci Pollut Res Int*, 30(44), 100072–100077. <https://doi.org/10.1007/s11356-023-29435-3>

Maryanaji, Z. (2020). The effect of climatic and geographical factors on breast cancer in Iran. *BMC Research Notes*, 13, 519. <https://doi.org/10.1186/s13104-020-05368-9>

Mufson, S. (n.d.). *Harvey causes chemical companies to release 1 million pounds of extra air pollutants - The Texas Tribune*. Retrieved <https://www.texastribune.org/2017/09/04/harvey-causes-chemical-companies-release-1-million-pounds-extra-air-po/>

Nagel, G., Stafoggia, M., Pedersen, M., Andersen, Z. J., Galassi, C., Munkenast, J., Jaensch, A., Sommar, J., Forsberg, B., Olsson, D., Oftedal, B., Krog, N. H., Aamodt, G., Pyko, A., Pershagen, G., Korek, M., Faire, U. De, Pedersen, N. L., Ostenson, C.-G., ... Weinmayr, G. (2018). Air pollution and incidence of cancers of the stomach and the upper aerodigestive tract in the European Study of the Cohorts for Air Pollution Effects (ESCAPE). *Int. J. Cancer*, 143, 1632–1643. <https://doi.org/10.1002/ijc.31564>

Naqvi, M., Gilani, S. Q., Syed, T., Marques, O., & Kim, H.-C. (2023). Skin Cancer Detection Using Deep Learning-A Review. *Diagnostics*, 13, 1–26. <https://doi.org/10.3390/diagnostics13111911>

Nogueira, L. M., Yabroff, R., & Bernstein, A. (2020). Climate Change and Cancer. *CA Cancer J Clin*, 70, 239–244. <https://doi.org/10.3322/caac.21610>

Pagano, C., Navarra, G., Coppola, L., Savarese, B., Avilia, G., Giarra, A., Pagano, G., Marano, A., Trifuoggi, M., Bifulco, M., & Laezza, C. (2023). Impacts of Environmental Pollution on Brain

Tumorigenesis. *Int. J. Mol. Sci.*, 24, 1–17. <https://doi.org/10.3390/ijms24055045>

Parent, M.-E., Goldberg, M. S., Crouse, D. L., Ross, N. A., Chen, H., Valois, M.-F., & Liautaud, A. (2013). Traffic-related air pollution and prostate cancer risk: a case-control study in Montreal, Canada. *Occup Environ Med*, 1–8. <https://doi.org/10.1136/oemed-2012-101211>

Parker, E. R. (2021). The influence of climate change on skin cancer incidence - A review of the evidence. *International Journal of Women's Dermatology*, 7, 17–27. <https://doi.org/10.1016/j.ijwd.2020.07.003>

Patil, V. S. (2023). Addressing the impact of the climate crisis on oral health. *Int J Prev Clin Dent Res*, 10, 20–22. https://doi.org/10.4103/ijpcdr.ijpcdr_6_23

PRESS RELEASE N° 221 IARC: *Outdoor air pollution a leading environmental cause of cancer deaths*. (2013). <http://www.iarc.fr/en/publications/books/sp161/index.php>

Puri, P., Nandar, S. K., Kathuria, S., & Ramesh, V. (2017). Effects of air pollution on the skin: A review. *Indian J Dermatol Venereol Leprol*, 83, 415–423. <https://doi.org/10.4103/0378-6323.199579>

Raaschou-Nielsen, O., Pedersen, M., Stafoggia, M., Weinmayr, G., Andersen, Z. J., Galassi, C., Sommar, J., Forsberg, B., Olsson, D., Oftedal, B., Krog, N. H., Aasvang, G. M., Pyko, A., Pershagen, G., Korek, M., Faire, U. De, Pedersen, N. L., Ostenson, C.-G., Fratiglioni, L., ... Hoek, G. (2017). Outdoor air pollution and risk for kidney parenchyma cancer in 14 European cohorts. *International Journal of Cancer*, 140, 1528–1537. <https://doi.org/10.1002/ijc.30587>

Rattray, N. J. W., Charkoftaki, G., Rattray, Z., Hansen, J. E., Vasiliou, V., & Johnson, C. H. (2017). Environmental influences in the etiology of colorectal cancer: the premise of metabolomics. *Curr Pharmacol Rep*, 3(3), 114–125. <https://doi.org/10.1007/s40495-017-0088-z>

Rodgers, K. M., Udesky, J. O., Rudel, R. A., & Brody, J. G. (2018). Environmental chemicals and breast cancer: An updated review of epidemiological literature informed by biological mechanisms. *Environmental Research*, 160, 152–182. <https://doi.org/10.1016/j.envres.2017.08.045>

Ruszkiewicz, J. A., Tinkov, A. A., Skalny, A. V., Siokas, V., Dardiotis, E., Tsatsakis, A., Bowman, A. B., Rocha, J. B. T. da, & Aschner, M. (2019). Brain diseases in changing climate. *Environ Res*, 177, 1–125. <https://doi.org/10.1016/j.envres.2019.108637>

Saad-Hussein, A., Ramadan, H. K.-A., Bareedy, A., & Elwakil, R. (2022). Role of Climate Change in Changing Hepatic Health Maps. *Current Environmental Health Reports*, 9, 299–314. <https://doi.org/10.1007/s40572-022-00352-w>

- Sadeghi, A., Leddin, D., & Malekzadeh, R. (2023). Mini Review: The Impact of Climate Change on Gastrointestinal Health. *Middle East J Dig Dis*, 15(2), 72–75. <https://doi.org/10.34172/mejdd.2023.325>
- Sakhvidi, M. J. Z., Lequy, E., Goldberg, M., & Jacquemin, B. (2020). Air pollution exposure and bladder, kidney and urinary tract cancer risk: A systematic review. *Environmental Pollution*, 267, 115328. <https://doi.org/10.1016/j.envpol.2020.115328>
- Scheurer, M. E., Danysh, H. E., Follen, M., & Lupo, P. J. (2014). Association of traffic-related hazardous air pollutants and cervical dysplasia in an urban multiethnic population: a cross-sectional study. *Environmental Health*, 13, 52, 1–8. <https://doi.org/10.1186/1476-069X-13-52>
- Schwerdtle, P. N., Bowen, K., & McMichael, C. (2018). The health impacts of climate-related migration. *BMC Med*, 16(1), 1–7. <https://doi.org/10.1186/s12916-017-0981-7>
- Silva, V. E. da. (2025). Air quality impacts and health effects (2025 article). *Environ. Res.: Health*, 3, 022003. <https://doi.org/10.1088/2752-5309/adcfb9>
- So, R., Chen, J., Mehta, A. J., Liu, S., Strak, M., Wolf, K., Hvidtfeldt, U. A., Rodopoulou, S., Stafoggia, M., Klompmaker, J. O., Samoli, E., Raaschou-Nielsen, O., Atkinson, R., Bauwelinck, M., Bellander, T., Boutron-Ruault, M.-C., Brandt, J., Brunekreef, B., Cesaroni, G., ... Andersen, Z. J. (2021). Long term exposure to air pollution and liver cancer incidence in six European cohorts. *Int. J. Cancer*, 149(11), 1887–1897. <https://doi.org/10.1002/ijc.33743>
- Terry, M. B., Michels, K. B., Brody, J. G., Byrne, C., Chen, S., Jerry, D. J., Malecki, K. M. C., Martin, M. B., Miller, R. L., Neuhausen, S. L., Silk, K., & Trentham-Dietz, A. (2019). Environmental exposures during windows of susceptibility for breast cancer: a framework for prevention research. *Breast Cancer Research*, 21(96), 1–16. <https://doi.org/10.1186/s13058-019-1168-2>
- Turner, M. C., Andersen, Z. J., & Baccarelli, A. (2020). Outdoor Air Pollution and Cancer: An Overview of the Current Evidence and Public Health Recommendations. *CA: A Cancer Journal for Clinicians*, 70. <https://doi.org/10.3322/caac.21632>
- US EPA, O. (n.d.). *Status of Superfund Sites in Areas Affected by Harvey*. Retrieved <https://www.epa.gov/newsreleases/status-superfund-sites-areas-affected-harvey>
- VoPham, T. (2019). Environmental risk factors for liver cancer and nonalcoholic fatty liver disease. *Curr Epidemiol Rep*, 6(1), 50–66. <https://doi.org/10.1007/s40471-019-0183-2>
- Wei, Y., Yazdi, M. D., Ma, T., Castro, E., Liu, C. S., Qiu, X., Healy, J., Vu, B. N., Wang, C., Shi, L., & Schwartz, J. (2023). Additive effects of 10-year exposures to PM2.5 and NO2 and primary cancer incidence in American older adults. *Environmental Epidemiology*, 7(4), 265–273. <https://doi.org/10.1097/EE9.0000000000000265>
- Wright, C. Y., Norval, M., Kapwata, T., Preez, D. J. du, Wernecke, B., Tod, B. M., & Visser, W. I. (2019). The Incidence of Skin Cancer in Relation to Climate Change in South Africa. *Atmosphere*, 10(10), 634. <https://doi.org/10.3390/atmos10100634>
- Yin, J., Wu, X., Li, S., Li, C., & Guo, Z. (2020). Impact of environmental factors on gastric cancer: A review of the scientific evidence, human prevention and adaptation. *Journal of Environmental Sciences*, 89, 65–79. <https://doi.org/10.1016/j.jes.2019.09.025>
- Youogo, L. M.-A. K., Parent, M.-E., Hystad, P., & Villeneuve, P. J. (2022). Ambient air pollution and prostate cancer risk in a population-based Canadian case-control study. *Environmental Epidemiology*, 6(219), 1–8. <https://doi.org/10.1097/EE9.0000000000000219>
- Yu, P., Xu, R., Yang, Z., Ye, T., Liu, Y., Li, S., Abramson, M. J., Kimlin, M., & Guo, Y. (2023). Cancer and Ongoing Climate Change: Who Are the Most Affected? *ACS Environ. Au*, 3, 5–11. <https://doi.org/10.1021/acsenvironau.2c00012>
- Yu, Q., Zhang, L., Hou, K., Li, J., Liu, S., Huang, K., & Cheng, Y. (2021). Relationship between Air Pollutant Exposure and Gynecologic Cancer Risk. *Int. J. Environ. Res. Public Health*, 18, 1–14. <https://doi.org/10.3390/ijerph18105353>
- Zhang, H.-W., Tsai, Z.-R., Kok, V. C., Peng, H.-C., Chen, Y.-H., Tsai, J. J. P., & Hsu, C. Y. (2022). Long-term ambient hydrocarbon exposure and incidence of urinary bladder cancer. *Scientific Reports*, 12, 20799. <https://doi.org/10.1038/s41598-022-25425-6>
- Zimta, A.-A., Gulei, D., Stefan, D. C., & Tomuleasa, C. (2021). The impact of climate change and land use on the cancer burden in the 21st century. *Climate and Land Use Impacts on Natural and Artificial Systems (Elsevier)*, 49–71. <https://doi.org/10.1016/B978-0-12-822184-6.00010-7>