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Air pollution and its impact on cancer incidence, cancer care and cancer outcomes

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ABSTRACT

Air pollution is an under-recognised global health threat linked to an increased risk of cancers and is due primarily to the burning of fossil fuels. This review provides a high-level overview of the associations between outdoor and indoor air pollution and cancer risk and outcomes.

Outdoor air pollutants are largely due to the burning of fossil fuels from human activities, although there is growing data implicating outdoor pollution from wildfire smoke. Indoor air pollution is primarily caused by burning solid fuel sources such as wood, coal and charcoal for household cooking and heating. There is a growing number of pieces of evidence linking exposure to pollution and the risk of developing cancers. The strongest evidence is seen on the positive association of air pollution, particularly particulate matter 2.5 with lung cancer. Emerging data implicate exposure to pollutants in the development of breast, gastrointestinal and other cancers. The mechanisms underlying these associations include oxidative stress, inflammation and direct DNA damage facilitated by pollutant absorption and distribution in the body.

References were identified through a PubMed search for articles published in 2000 to October 2024 using the terms 'air pollution' or 'pollutants' and 'carcinoma' or ''cancer'.

Air pollution poses significant risks to health. Its health impacts, including cancer risks, are often underestimated. Hazardous pollutants have been studied in several epidemiological cohort studies. Despite the mounting evidence, air pollution is often overlooked in predictive cancer risk models and public health intervention.

INTRODUCTION

Air pollution is a global health issue. It has a significant effect on public and individual health. It has been associated with many non-communicable diseases including asthma, birth defects in children, cancer, cardiovascular and cerebrovascular disease, diabetes, neurodevelopmental disorders and respiratory diseases.[1 2] According to the WHO, 99% of the world's population resides in areas where the air quality exceeds the recommended guidelines.[3] Globally, 7.3 billion people are directly exposed to polluted air.[4] The purpose of this narrative review is to provide a high-level overview of the existing literature on outdoor and indoor air pollution and various hazardous pollutants. The goal is to review the current available evidence on the relationship between air pollution and different types of cancer, and to educate the reader as to its effects on cancer incidence, cancer care and cancer outcomes.

It should be noted that air pollution is a mixture of many different components, and oftentimes, it is difficult to single out one specific pollutant or even one component of particulate matter (PM) as being solely responsible for the observed cancer risks. For example, while PM2.5 is strongly associated with lung cancer risk, it is important to note that air pollution consists not only of PM2.5, but rather is a complex mixture of various components, including elemental carbon, organic carbon, nitrates, sulfates and metals. Each of these components may have different mechanisms of action, and their relative contributions to cancer risk may vary depending on the source of the pollution, geographical region and other environmental factors. This makes it particularly challenging to pinpoint one specific component or pollutant as the primary driver of cancer risk.

In light of this, while we have highlighted the key pollutants that have been consistently associated with cancer risk, we emphasise the need for a broader approach to understanding the health impacts of air pollution. We believe that a comprehensive understanding of the entire air pollution mixture, while attempting to focus on individual components, is crucial for developing effective public health strategies.

Outdoor air pollution

Exposure to air pollution is estimated to be responsible for 8.9 million deaths in 2015.[5] Major components of air pollution are primary air pollutants that are directly emitted into the atmosphere from identifiable sources, such as the burning of fossil fuels

in combustion engines, factories, healthcare, agriculture and electricity generation. These include PM, especially those with diameter<10 µm and<2.5 µm $(PM)_{2.5}$, sulphur oxides (SOx), nitrogen oxides (NO_x), carbon monoxide (CO) and volatile organic compounds (VOCs). VOCs are a group of organic chemicals that play a significant role in air pollution. Formaldehyde is a VOC that is emitted from vehicle exhaust, industrial processes and wood-burning, and is a known carcinogen.[6] It is one of the most important hazardous air pollutants (HAPs) that is associated with health risks, accounting for over 50% of the total HAPs-related cancer risks in the USA.[7] PM2.5 is composed of inorganic ions, organic compounds, toxic metals and mineral dust particles; as mentioned above, it is difficult to single out one component of PM2.5 as being solely responsible for the observed cancer risks. Secondary air pollutants include gaseous ozone, a major component of photochemical smog, and PM2.5, which are formed in the atmosphere from primary pollutants.

Outdoor air pollutants come primarily from the burning of fossil fuels from human activities, such as from the use of industrial machinery, power-producing stations, combustion engines and cars. End use of processed and unprocessed oil and natural gas in fuel combustion and industrial processes contributes significant emissions of air pollutants.[8] Rapid urbanisation and population growth lead to scale expansion of cities, and consequently an increase in the number of motor vehicles. Traffic-related pollutants have become the main source of pollution and PM2.5 in big cities.[9–11] Other human activities are contributing to pollution to a lesser extent, such as field cultivation techniques, gas stations and fuel tank heaters. A natural source of air pollution, unrelated directly to the humans burning of fossil fuels, are forest fires, which are becoming more common and are fuelled by climate change. Other sources of air pollution such as sand comprise a very small proportion of air pollution except in arid and semi-arid countries.

Long-term exposure to PM2.5 from coal combustion is associated with increased mortality from lung cancer. Arsenic, selenium and sulphur are components most closely associated with coal combustion. These were also significantly linked to lung cancer mortality.[12]

Indoor air pollution

Indoor air pollution is primarily caused by burning solid fuel sources such as wood, crop waste, coal, charcoal and dung for household cooking and heating. Wood smoke is a complex mixture consisting of PM, various gases and chemicals. Some have been classified as hazardous pollutants and carcinogens. Fumes and PM generated from burning cooking oils and biomass contribute to indoor air pollution in homes across the world, particularly in developing nations, parts of Africa and Asia. In poorly ventilated homes, smoke can have much higher levels of fine particles worsening indoor air quality. Biomass burning emits air pollutants including varying sizes of PM, VOCs, polycyclic aromatic hydrocarbons (PAHs), CO and benzo[a]pyrene (B[a]P).[13] Indoor air can be heavily polluted with B[a]P, a PAH produced during the burning of fossil fuels, wood and other organic materials. This is especially prevalent in less developed countries, where biomass burning is a common practice. High levels of indoor B[a]P have been found in kitchens during cooking, in buildings with poor ventilation, and in workplaces such as aluminium factories and coke-oven facilities.[14]

Benzene, a known carcinogen, is an aromatic hydrocarbon that occurs naturally in crude oil, and is a byproduct of several industrial processes.[15] It can be produced through the distillation of coal, the refining of petroleum and the combustion of organic materials. Additionally, benzene is used in the manufacture of numerous chemicals such as plastics, synthetic fibres, dyes and rubber.[15]

A variety of VOCs are emitted from modern household products such as paints, lacquers, cleaning liquids and furnishings. Most of the risks from VOCs, including chloroform, formaldehyde and naphthalene originate from indoor sources.[16] Those emitted from household products include non-methane hydrocarbons, benzene and toluene. The US Environmental Protection Agency (EPA) evaluated the nationwide risk associated with outdoor concentrations of most HAPs. The EPA found that nearly half of the total estimated lifetime cancer cases from HAPs could be attributed to VOCs, with an additional 40% linked to PAHs.[17]

Air pollution and climate change

Climate change is caused by increasing greenhouse gases (GHGs) in the atmosphere, such as methane, CO2, nitrous oxide and hydrofluorocarbons, among others. These GHGs have the ability to trap the sun's heat, and come primarily from burning fossil fuels which releases carbon that was previously stored in the ground as coal, unrefined oil or gas.

A 'natural' source of air pollution is that produced by wildfires. As the earth warms, and heat waves and droughts are becoming more common, wildfires are becoming more frequent, destructive and intense. Although wildfire smoke is not specifically classified as a Group 1 carcinogen, certain components of wildfire smoke are recognised as carcinogenic, including PM and PAHs. Levels of wildfire PM2.5 can greatly exceed those of ambient PM2.5, spiking episodically within a short period of time even just a few hours after the onset of a wildfire.[18]

Recent studies[18 19] suggest that wildfire PM has higher toxicity than equal doses of ambient PM2.5.

Wildfire PM has 5–20% elemental carbon and at least 50% organic carbon.[19] Elemental carbon is pure carbon in the form of soot and is produced during the incomplete combustion of carbon-containing materials. Organic carbon consists of a variety of carbon-containing compounds produced through the pyrolysis and combustion of biomass. These compounds in wildfire smoke generate more free radicals, increasing their potential to cause inflammation and oxidative stress in the lungs.[18]

The smoke from wildfires can spread thousands of miles; smoke from wildfires on the West Coast of the USA or Canada can impact the East Coast.

Air pollution and cancer

Lung cancer

The International Agency for Research on Cancer (IARC) has classified PM, diesel exhaust and outdoor air pollution overall as Group 1 carcinogens.[20] There is incontrovertible evidence that long-term exposure to air pollution is associated with the development of lung cancer. The overwhelming prevalence of tobacco smoking as a contributor to lung cancer may have diverted focus from recognising outdoor air pollution as another preventable factor

Pathogenesis

Burning of fossil fuels results in submicron combustion-related PM containing numerous toxic compounds including acids and heavy metals and can penetrate deeper into the lung than the larger PM. PM induces oxidative stress in epithelial cells, generating reactive oxygen species that may damage DNA, proteins and lipids.[21]

Lung cancer in never-smokers is frequently adenocarcinomas with oncogenic epidermal growth factor receptor (*EGFR*) mutations. Hill *et al*[22] explored how PM drives and promotes lung cancer. PM2.5 induces sustained inflammation with recruitment of macrophages into the lungs. The macrophages provide signalling molecules, including interleukin (IL)-1β, which can activate some lung stem cells called alveolar type 2 cells to start to grow, particularly if these cells have pre-existing *EGFR* mutations. The study found an association between the frequency of *EGFR* mutant lung cancer incidence, IL-1 beta levels and increasing PM2.5 levels. Temporal analysis demonstrated that 3 years of exposure to PM2.5 may be enough to increase the risk of *EGFR*-driven lung cancer.[22]

The compounds in wildfire smoke tend to generate more free radicals than ambient air pollution, and therefore have more potential to cause inflammation and oxidative stress in the lung.[23]

Epidemiological studies

Components of traffic-related air pollution have been associated with lung cancer incidence, including nitrogen oxide (HR, 1.15 per 50 parts per billion (ppb); 95% CI, 0.99 to 1.33), NO_2 (HR, 1.12 per 20 ppb; 95% CI, 0.95 to 1.32), PM2.5 (HR, 1.20 per 10 µg/m^3; 95% CI, 1.01 to 1.43), CO (HR, 1.29 per 1000 ppb; 95% CI, 0.99 to 1.67) and regional benzene (HR, 1.17 per 1 ppb; 95% CI, 1.02 to 1.34) exposures.[24] These findings in a large multiethnic population reflect an association between lung cancer and the mixture of traffic-related air pollution and not a particular individual pollutant.[24]

There is significant evidence linking PM2.5 with lung cancer mortality. Pope *et al*[25] provided the first evidence on the association between long-term exposure to fine particulate air pollution and lung cancer mortality. Based on a large cohort and extended follow-up, each 10 µg/m^3 elevation in fine particulate air pollution was associated with approximately 8% increased risk of lung cancer mortality. A meta-analysis by Hamra *et al*[26] included findings from 14 studies of outdoor air pollution conducted mostly in North America and Europe. It reported a statistically significant 9% increase in risk for lung cancer incidence or mortality per each 10 µg/m^3 increase in PM2.5 concentrations. A more recent updated meta-analysis[27] of findings from 20 cohort studies reported a 14% increase in lung cancer incidence or mortality per 10 µg/m^3 of PM2.5. Other meta-analyses have also confirmed the association between air pollution exposure and lung cancer incidence and mortality.[28–31] The strength of association for cancer incidence was higher in males than females, which could be attributed to higher rates of smoked tobacco use in males.[31]

Smoke from wildfires has also been linked to cancer. A population-based observational cohort study involving 2 million people for a median of 20 years studied long-term exposure to wildfires and cancer incidence in Canada. They found that when compared with unexposed populations, cohort members exposed to a wildfire within 50 km of residential locations in the past 10 years had a 4.9% relatively higher incidence of lung cancer and a 10% relatively higher incidence of brain cancers.[32] Exposure to wildfire smoke while recovering from lung cancer surgery was associated with an increased risk of dying: a 43% higher risk if the time from surgery to wildfire smoke was between 0 and 3 months; 39% if it was between 4 and 6 months and 17% if between 7 and 12 months.[33]

The US-based prospective Sister Study[34] followed 50 226 women without a prior lung cancer diagnosis to determine whether exposure to indoor wood smoke from fireplaces and stoves is associated with incident lung cancer. The women were followed for an average of 11.3 years; 347 medically confirmed lung cancer cases were reported. Compared with those without a wood-burning fireplace/stove, women who used their wood-burning fireplace/stove≥30 days/year had an elevated rate of lung cancer (HR adjusted (HR adj)=1.68; 95% CI, 1.27 to 2.20).[34] Associations were elevated across all income groups and among those who lived in urban or rural and small-town settings.[34]

Diesel exhaust has been labelled a Class 1 carcinogen by the IARC. Based on data from three occupational cohorts,[35 36] exposure-response estimates for diesel engine exhaust and lung cancer mortality estimated that approximately 6% of annual lung cancer deaths may be due to exposure to diesel engine exhaust. In the Diesel Exhaust in Miners Study,[35] a cohort mortality study of 12 315 workers exposed to diesel exhaust at eight US non-metal mining facilities found that higher than standardised mortality ratios for lung cancer (1.26, 95% CI, 1.09 to 1.44), oesophageal cancer (1.83, 95% CI, 1.16 to 2.75) and pneumoconiosis (12.20, 95% CI, 6.82 to 20.12)

were elevated compared with state-based mortality rates. A nested case–control study of the same population[36] found increasing trends in lung cancer risk with increasing cumulative respirable elemental carbon intensity and average respirable elemental carbon intensity. Among heavily exposed workers (ie, above the median of the top quartile), risk was approximately three times greater (OR=3.20, 95% CI, 1.33 to 7.69) than that among workers in the lowest quartile of exposure.[36]

Never-smokers

Long-term exposure to air pollution causes lung cancer even in people who have never smoked. One study found lung cancer mortality was adversely associated with increases in PM2.5, in both the overall population studied as well as in a cohort of over 340 000 never smokers. The risk of all cancer mortality was adversely associated with a 19% increase per 10 µg/m^3 increase in PM 2.5 in never-smokers.[37] Several recent meta-analyses have reported that the relative risk of lung cancer ranges from about 1.18–1.24 for a 10 µg/m^3 increase in PM2.5 level in smokers, and 1.15–1.18 in never smokers.[26 38 39] Aggregate findings from meta-analyses by Huang *et al* and Ramamoorthy *et al*[28 31] indicated that former smokers might have a higher lung cancer risk linked to PM2.5 compared with current smokers and never smokers across mortality and incidence.

Epidemiological studies in low- and middle-income countries (LMICs) have found an association between household wood combustion and lung cancer. The Sister Study[34] found that increased frequency of using wood-burning in indoor fireplace and stove was associated with incident lung cancer even in never smokers, which was related to their use (1–29 days/year HR adj=1.64; 95% CI, 0.87 to 3.10) and≥30 days/year (HR adj=1.99; 95% CI, 1.02 to 3.89).

Indoor air pollution

Many Asian countries have traditional cooking practices that involve heating cooking oils to very high temperatures, especially when frying. The incidence of lung cancer was noted to be higher with deep frying than stir-frying and in homes with poor ventilation.[40–42] A large retrospective cohort study among domestic coal users in China[43] reported that the absolute risk of death from lung cancer before 70 years of age was 18% and 20% for men and women, respectively.

There is increasing evidence that air pollution is not limited to lung cancer, but also cancer of other sites. This could be due to exposure through absorption, metabolism and distribution of inhaled carcinogens released as primary PM emissions or bound to particles, including PAHs, heavy metals and other VOCs.

Breast cancer

A growing number of epidemiological studies have suggested that exposure to ambient air pollution may play an important role in breast cancer development. Air pollutants can potentially influence the occurrence of breast cancer by increasing breast density, which is a known risk factor.[44] PM concentration has been positively correlated with breast tumour size and breast cancer aggressiveness. Air pollutants can reach breast tissue; one study found some inhaled toxic substances present in breast fluid.[45]

A recent study[46] on PM and breast cancer incidence was on a large, prospective cohort of women (n=196 905) across the USA. There was an 8% increase in breast cancer risk for a 10 µg/m^3 increase in estimated historical exposure to ambient PM2.5 concentrations during a 10–15 year-period before study enrolment. This association was evident for oestrogen receptor–positive tumours and not for oestrogen receptor–negative tumours.

The adverse effects of PM on mortality may not only occur in lung cancer, but also in breast cancer. A meta-analysis by Zhang *et al*[47] demonstrated the association between PM exposure and breast cancer morbidity and mortality. It showed that both PM2.5 and PM10 are associated with a significantly increased risk of breast cancer mortality, especially in the subgroup of high exposure level. Some potential pathways may involve inflammation due to oxidative stress.

NO2 and other nitrous oxides have also been associated with increased breast cancer incidence.[48 49] Traffic is the major contributor to NO2 concentrations. As motor-vehicle traffic is generally the main emission source of NO2, exposure to NO2 can be considered as a surrogate of exposure to traffic-related air pollution (TRAP). A large nested case–control study[50] investigated long-term exposure to NO2 over up to 22 years and its association with breast cancer risk. The long-term exposure to ambient air NO2 was positively associated with breast cancer risk overall. Subgroup analyses showed NO2 being positively associated with postmenopausal, hormone receptor-positive and ductal-lobular breast cancer. The results strengthen the evidence on the association between NO2 exposure and breast cancer risk.

A recent meta-analysis reported an association between increasing NO2 exposure of 10 µg/m^3 and a 3% increased risk of breast cancer, but no significant association with PM2.5 and PM10 exposure was found.[51] A systematic review of observational studies[52] assessed the exposure to TRAP and breast cancer risk. Although there was limited evidence of an association for TRAP, the review showed a significant association between NO2 exposure and breast cancer risk, thus providing additional support for air pollution carcinogenicity.

PAHs originating from traffic emissions may cause breast cancer through DNA damage, aberrant DNA methylation and oestrogenic and antioestrogenic activities. PAHs are recognised to be endocrine-disrupting chemicals with specific implications for breast and prostate cancer. In 2022, the first meta-analysis on the association between PAH exposure and breast cancer found a significant association between the two.[53]

Indoor wood-burning stove and fireplace use was also associated with an increased risk of breast cancer. The Sister Study[34] recruited 50 884 participants from 2003 to 2009. Participants who were breast cancer-free women in the USA or Puerto Rico with a sister with breast cancer were eligible. A total of 2416 breast cancer cases were diagnosed during follow-up (mean=6.4 years). Having an indoor wood-burning stove/fireplace in the longest adult residence was associated with a higher breast cancer risk (HR=1.11 (95% CI, 1.01 to 1.22)). The increased risk for weekly wood-burning stove/fireplace use was observed for invasive breast cancer (n=265; HR=1.25 (95% CI, 1.07 to 1.46)] but not ductal carcinoma in situ.

GI malignancies

Small particles readily absorbed in the lungs following inhalation can be delivered through the bloodstream, then get deposited in other body tissues including the gut.[54] PM reaching the bronchioles and alveolar spaces may be phagocytosed by alveolar macrophages,[55] then sequestered and trapped in the airway by a protective mucus layer.[56] The trapped particles are propelled by cilia through the oropharynx and into the gastrointestinal (GI) tract through mucociliary clearance.[57] Mucociliary transport of PM inhaled in the lungs and then cleared into the upper GI tract has been demonstrated in human studies of non-smokers.[58]

A systematic review and meta-analysis by Pritchett *et al*[59] assessed whether exposure to outdoor PM was associated with GI cancers. The results from the meta-analysis indicate that PM exposure may be associated with mortality or incidence for some GI cancers, particularly colorectal and liver. Fu *et al* [60] found a strong association between PM exposure and increased colorectal cancer (CRC) risk, identifying 10 eligible studies across multiple countries and regions in North America and Asia. It revealed an association between PM and increased risk of CRC and mortality. The elevated risks of CRC associated with PM were different across countries and regions. Incidence and mortality risks were noted to be higher in North America than in Asia.

A comprehensive systematic review[61] studied the association between air pollution and liver cancer. A total of 13 cohort studies were obtained with 10 961 717 participants. PM2.5 was the most frequently examined pollutant, then followed by NO2 and NO_x. PM2.5 was associated with liver cancer mortality. There was no association with other air pollutants. So, *et al*.[62] examined the association between long-term exposure to air pollution and liver cancer incidence in a pooled six European cohorts over a mean follow-up of 18 years. It identified relevant sources by analysing specific elements of PM2.5. There were detected associations between long-term exposure to NO2, PM2.5 and black carbon and liver cancer incidence. Strongest associations were found for sulphur and vanadium components of PM2.5.

Although there have been case–control studies that showed a positive association between burning biomass fuels and GI cancers, specifically oesophageal cancer[63–67] and gastric cancer,[68 69] the Golestan Cohort Study[70] is the first large population-based prospective study that provided strong evidence on the higher risk of developing colon, oesophageal and gastric cancers with use of biomass fuels for household cooking and heating. The study, which followed up 50 045 participants for more than 10 years, also found out that use of kerosene for household cooking and heating is associated with a higher risk of developing oesophageal cancer.[70] The study adds to the evidence that increased cancer risk among individuals who are exposed to the indoor combustion of biomass and kerosene fuels may extend beyond the respiratory system and affect several sites of the gastrointestinal tract.[70] The exact mechanisms that increase these risks are still not known. However, one possibility might be through local and systemic exposure of digestive organs to the carcinogenic compounds present in the emissions from combustion of solid fuels and kerosene.[71]

Brain tumours

Inhalation of pollutants may result in the release of local inflammatory mediators of the lung, resulting in chronic inflammation, oxidative stress and DNA damage.[72] Cytokines, such as interleukins (IL-6, IL-8) and tumour necrosis factor, induced by systemic inflammation, can cross the blood-brain barrier and may lead to activation of microglia, which may induce a tumour microenvironment.[73] Ultra-fine particles from incomplete combustion of fossil fuels in motor vehicles can enter the brain by crossing the blood-brain barrier[72] or through the nasal passages, olfactory neurons and then accumulate in the brain.[74 75] PM2.5 can also disturb the brain-blood barrier by causing oxidative stress and neuritis.[76]

Shen *et al*[77] conducted a comprehensive meta-analysis to explore the connection between common air pollution particles and adult brain tumours. Their findings revealed a significant link between exposure to black carbon (BC), usually referring to elemental carbon,[78] NO_x and brain tumours in adults, highlighting the detrimental effects of certain air pollutants on brain health. Building on these findings, Hvidtfeldt *et al*[79] conducted a pooled analysis of six European cohorts comprising 302 493 individuals to further investigate the association between residential exposure to air pollutants and malignant intracranial central nervous system (CNS) tumours. Their results indicated a suggestive association between NO2, PM2.5, BC and CNS tumour incidence, reaffirming the role of certain air pollutants in brain tumour development.[79] Similarly, Hassanipour *et al*[76] conducted a systematic review and meta-analysis focusing on the impact of long-term exposure to ambient air pollution on brain tumour incidence in adults. Their analysis identified ozone and PM2.5 absorbance as significant contributors to brain tumour development. Exposure to ozone, PM2.5 absorbance, proximity to traffic, NO_x incidence index and ozone were statistically significant.

A population-based observational cohort study studied long-term exposure to wildfires and cancer incidence in Canada. Following 2 million people for a median of 20 years, they found that when compared with unexposed populations, cohort members exposed to a wildfire within 50 km of residential locations in the past 10 years had a 10% relatively higher incidence of brain cancers.[32]

Ovarian cancer

$PM_{2.5}$ contain PAHs and derivatives that are associated with genetic polymorphisms in the activation of certain carcinogens[80] and steroid hormone metabolism, thereby promoting the proliferation of cancer cells.[81] The possible mechanisms are oestrogen-like effects and genetic mutations. One review[82] on the role of air pollution in ovarian cancer risk included studies on the types of measured ambient air pollutants and traffic indicators for air pollution. 15 of the studies included in the review showed a positive correlation between ambient air pollution exposure and an increase in ovarian cancer risks.[82]

Childhood cancers and leukaemia

The most frequent cancer among children is leukaemia, followed by brain and other CNS tumours. Although important progress has been achieved regarding cure rates for paediatric cancers, the causes of childhood cancers are still not clear. Several epidemiological studies suggested that environmental pollution may have a role in aetiology. Prenatal and postnatal exposure to environmental contaminants, including air pollution, electromagnetic fields, pesticides, parental smoking and radon, have been studied worldwide and are proposed as childhood cancer risk factors.[83–85]

A wide number of studies evaluated outdoor air pollution, specifically exposure to contaminants released by motorised traffic[86–88] and childhood cancer risk. Studies have associated an increased risk of CNS cancers, leukaemia, neuroblastoma, Wilms tumour and bone cancer[89–96] with living near heavy-traffic roads or proximity to industrial and urban sites. Childhood leukaemia may occur due to chromosomal translocation of a preleukaemic clone and may be triggered by exposure to environmental factors such as benzene.[97]

Benzene is a major component of air pollution, and it induces genotoxic and non-genotoxic events in utero. A meta-analysis by Wei *et al*[98] examined the association between the exposure to PM, benzene, NO2 and NO_x, and leukaemia risk, and showed that the highest exposure to benzene and NO_x were positively correlated with leukaemia risk when compared with the lowest exposure categories for each air pollutant. Exposure to benzene in the third trimester of pregnancy, as well as exposure to NO_x in the second trimester and entire pregnancy, could also increase the risk of leukaemia.

Collectively, these studies underscore the importance of understanding the impact of air pollution on cancer. While specific air pollutants such as BC, NOx, ozone and PM2.5 absorbance have been consistently associated with an increased risk of cancer, further research is needed to elucidate the underlying mechanisms and inform targeted interventions to mitigate the adverse effects of air pollution on health.

UNDER-RECOGNISED RISK OF AIR POLLUTION: PETROLEUM

Workers in the petroleum industry are exposed to known or potentially harmful substances.[99] Emissions from oil and gas extractions are among the major sources of air pollution in areas and communities where those facilities are located and operate.[100–102] Potential harmful exposures include heavy metals and hydrocarbons such as benzene.[103]

A review focusing on haematological malignancy among residents near petroleum industry sites and facilities has also found elevated risks for leukaemia.[104] Schnatter *et al*[105] assessed the risk of 11 cancers among petroleum refinery workers. There were elevated risks of mesothelioma, malignant skin melanoma and acute lymphoid leukaemia.[106] Other studies[107–110] showed that petroleum workers had elevated risks for mesothelioma, multiple myeloma, skin melanoma, prostate and urinary bladder cancer. Asbestos, which causes mesothelioma and cancers of the lung, larynx and ovary, has been used extensively in petroleum industries.[111 112]

IMPACT OF AIR POLLUTION ON UNDER-REPRESENTED MINORITIES AND MARGINALISED COMMUNITIES

Climate change exacerbates cancer disparities.[113 114] In the USA, PM 2.5 polluters disproportionately and systemically affect people of colour.[115] LMICs that are least responsible for GHG emissions are the most threatened by the multiple risks of global warming and lack the resources to resist the forces of climate change and survive. Communities of colour, indigenous peoples, the poor and low- and middle-income communities that are most vulnerable to rapidly changing climate conditions have the fewest resources to prepare for and recover from extreme weather events and other climate-related hazards.[116]

Pollution in LMICs caused by industrial emissions, toxic chemicals and vehicular exhaust has been overlooked in both the international development and the global health agendas. Several communities in LMICs have substantial occupational or residential exposures from high crude oil production capacity and artisanal refining activities. Angola and Nigeria are top oil producers. However, there have been no studies done on petroleum workers and residents living in oil-producing communities in Africa. There have been frequent oil spills that contaminate landscape and pollute soil and ground water. There has also been constant emission of hydrocarbons and other toxic chemicals in the Niger Delta region of Nigeria.[117] In the same region, houses are situated near petroleum oil wells with pipelines running across various communities. Crude oil is sometimes used for dermal care and ingested

for childhood convulsion treatments.[118] The United Nations Environment Programme reported that benzene level was 900 times above WHO guidelines in some spots in Ogoniland, Niger Delta region of Nigeria.[119] Major oil pollution has also been reported in some other petroleum-producing regions of the world such as the Middle East and the Russian Federation.[120 121]

CHALLENGES AND BARRIERS

Air pollution is a complex mixture of pollutants, including PM, VOCs, NO and heavy metals, with varying sources and compositions. Assessing individual exposure levels and incorporating these data into predictive risk models can be challenging due to the lack of precise exposure measurements and standardised methods for quantifying exposure over time. There are challenges in effectively comparing the impact of PM2.5 from specific sources, hence would need long-term research studies to quantify cancer risk and source-specific PM2.5.

Addressing these challenges and barriers requires interdisciplinary collaboration among researchers, healthcare providers, policymakers and environmental experts to improve risk assessment methodologies, develop novel biomarkers for early detection, enhance surveillance systems for environmental exposures and implement targeted screening and intervention strategies tailored to the unique needs of populations at risk of air pollution-related cancer.

We are likely to see increasing cases of cancer in the future, given the significant latent time between exposure to air pollution and the development of cancer. The incidence of lung cancer in the future will likely be further increased and probably occur more frequently in younger patients with less smoking history. Pollution levels remain unabated, and in some situations continue to rise.

RESEARCH GAPS

The impact of air pollution on human health has garnered increasing attention in recent years, but there are still significant gaps to be addressed. Despite growing evidence linking air pollution to cancer risk, there are still gaps in understanding the dose-response relationship, synergistic effects with other risk factors and long-term health implications of chronic exposure. Limited data availability and inconsistent methodologies in epidemiological studies hinder the integration of air pollution data into predictive risk models. The current widely used LDCT (Low Dose CT) screening based on cigarette exposure does not reflect the carcinogenic risk of pollution exposure in most of the global population and will need to be modified in the future. Additional studies are also needed to clarify the relationship between air pollution and other types of cancer.

Healthcare represents about 8% of the USA's carbon footprint (the total amount of GHGs produced). More research is needed as to how to make these systems more resilient. Life cycle analysis on chemotherapy and other commonly used agents needs to be conducted to determine which products are safe, effective and can lower healthcare's carbon footprint.

There is a need for more focused studies in under-researched areas. It is important to conduct well-designed epidemiological studies on workers and residents with industrial and occupational exposures of high petroleum production, especially in low-income countries. There is much opportunity to know and understand the cumulative impacts of environmental stressors, long-term effects of low-level air pollution exposure, as well as on the synergistic effect of multiple air pollutants. Despite the major advances in knowledge surrounding associations of outdoor air pollution and lung cancer, additional questions remain, including the role of PM during the cancer continuum.

CONCLUSION

Outdoor air pollutants are largely due to the burning of fossil fuels from human activities, although there is growing data implicating outdoor pollution from wildfire smoke with cancer. Indoor air pollution is primarily caused by burning solid fuel sources such as wood, coal and charcoal for household cooking and heating. Large epidemiological cohort studies have linked PM2.5 to lung cancer, although emerging data implicate exposure to pollutants in the development of breast, gastrointestinal, brain, leukaemia and childhood cancers. The mechanisms underlying these associations include oxidative stress, inflammation and direct DNA damage facilitated by pollutant absorption and distribution in the body.

Air pollution and its components are the most widespread environmental carcinogens—they endanger the health of billions. In addition to being a global burden of disease and cause of widespread disability and premature death, air pollution has major impacts on cancer incidence, care and outcomes. Despite its substantial effects on human health, economy and environment, air pollution has been neglected, most especially in LMICs.

Improving air quality will reduce the incidence and outcomes of diseases including cancer. Future research should continue to investigate the links and underlying causes between poor air quality, lung disease and cancer. Ultimately, improving air quality is a complicated process that demands a multifaceted approach and tailored solutions to be effective.

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