

# The Environmental Causes of Cancer: A Literature Review

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Premal PATEL\* <sup>1</sup>, James GOMES <sup>2</sup>

<sup>1</sup> Student, University of Ottawa, Canada

<sup>2</sup> Professor, University of Ottawa, Canada

\* *Auteur(e) correspondant* | *Corresponding author*: N/A

## Abstract:

Social media holds considerable potential for health promotion and other health intervention activities, as it addresses some of the limitations in traditional health communication by increasing accessibility, interaction, engagement, empowerment and customization. The use of social media increases the potential for easy access to preventive medicine, interaction with health care providers, interprofessional communication in emergency management, and public health. However, more research is needed to determine its long term effectiveness and to maximize the strategic presence of health organizations on social networking websites. This paper provides encouraging information about the possibilities of using social media to improve access to health information and health care providers, as well as to promote positive health behaviour change. It is essential for health promotion organizations to capitalize on the opportunities provided by social media, in order to modernize strategies to reach all age groups and to tailor programs to current communication trends, all of which are offered at a relatively low cost.

## Keywords:

Social media, social networking, health promotion, health communication, online health information, emergency management, interprofessional communication

## Introduction

Cancer is a multifactorial disease with a long latency and except for a few cancers the risk factors remain to be identified and the etiological relationship remains to be explored. Risk factors can include lifestyle, dietary habits, infections, environmental contaminants, pollution, occupational hazards, and others (Boffetta & Nyberg, 2003). Environmental factors are also believed to risk factors; however, these hazards have been largely overlooked until recently. The difficulty lies in the fact that there are many contaminants in the environment which can influence our health and wellbeing, and their impact on health remains to be documented. Furthermore, the definition of environment is still debatable; some arguing for the broad approach and others suggesting the narrow approach. Some definitions include lifestyle and dietary habits whereas others use a strict definition of contaminants outside the human body. These differences in the definitions change the environmental attributable fraction of cancer.

The World Health Organization (WHO) has stated that roughly 19% (12%–29%) of all cancers are attributable to the environment, which is equivalent to 1.3 million deaths each year (WHO, 2006). In the developed countries, it was estimated that 16% (10–34%) of cancers in men (other than lung cancers), and 13% (10–23%) in women, are attributable to the environment (WHO, 2006). In the developing countries, the corresponding attributable fractions are 18% (10–45%) in men and 16% (10–35%) in women. The uncertainty surrounding these estimates is due to the fact that evidence linking specific environmental and occupational exposures to various cancers is incomplete (WHO, 2006).

In developing countries, the attributable environmental fractions were 33% (6–65%) for men, and 25% (6–37%) for women (WHO, 2006). It was estimated that environmental factors account for 31% of the global disease burden of lung cancer and 30% (6–55%) of the disease burden in developed countries, for both men and women (WHO, 2006). Other studies assessing the environmental attributable fraction of cancer have reported lower estimates. Health Canada estimates that only 10–15% of cancers are linked the environment (Boyd & Genius, 2008). Whereas, other studies have shown the environmental fraction in Canada to be 5–15%, however, this is due to a narrower definition of environmental risk factor than the WHO (Boyd & Genius, 2008). It was estimated that 15.6% of the worldwide incidence of cancer in 1990 could be attributed to in-

fection, however, the range is quite disperse as 10% of cancer is attributable infections in high income countries whereas up to 25% in Africa (Belpomme et al., 2007).

The purpose of this review is to examine and evaluate the scientific literature on environmental cancers and identify potential environmental risk factors and calculate environmental attributable fraction of human cancer. This study reviews the environmental causes of cancer.

## Methodology

The first task in this study was to examine the different definitions of the environment. The working definition adopted by several agencies was searched including the Canadian Cancer Society, National Institute of Health Sciences, International Agency on the Research of Cancer (IARC), Environmental Protection Agency (EPA) and the World Health Organization (WHO).

### *Broad Definition*

The National Institute of Health Sciences uses a broad definition that incorporates lifestyle factors, dietary habits, exposure to agents in the ambient environment and in the workplace (Cancer and the Environment, 2003).

### *Narrow Definition*

The WHO applies a narrow definition to the term “environment”. This includes only the physical, chemical and biological factors that are external to the human host, and all related behaviors, but excluding those natural environments that cannot reasonably be modified (Prüss-Üstün & Corvalán, 2006). For the purposes of this study we have adopted the WHO definition of “environment”.

The following databases were searched for this study: Pubmed, Embase, Scopus, and Toxline. The search terms used to retrieve the articles were “environmental cancers, attributable fraction, environment and cancer, environmental burden of cancer”. All articles were then collated and compiled in Refworks. The articles were examined for compatibility with the inclusion and exclusion criteria. All the articles were assessed by reading the title, abstract and the body of the text. Data abstraction was carried out for all

the articles selected for inclusion in this review.

### Inclusion Criteria

The following inclusion criteria were used to screen potential studies using the article titles and abstracts:

1. Review articles and original research articles were selected.
2. The term environment defined by the WHO was considered as relevant. Therefore articles focusing on indoor and outdoor air contaminants, contamination of water and food, electromagnetic fields were included
3. Articles on environmental causes of cancer were also included.

### Exclusion Criteria

The following exclusion criteria were applied to screen articles that were not relevant for this review:

1. All studies that did not focus on environment and cancer.
2. The definition of the term “environment” that did not include the WHO terms of reference.
3. Studies that did not describe the etiological relationship between environmental factors and cancer.

### Results & Discussion

A number of cancers were identified as etiologically associated with environmentally related risk factors. A list of these cancers and the related etiological agent is shown in Table 1. A narrative of this relationship between cancer and

**Table 1** Environmental cancers and associated etiological agents

Cancer	Environmental Agent	Cancer	Environmental Agent
Bladder Cancer	Environmental exposure to arsenic. (Boyd & Genius, 2008) Arsenic oxides if inhaled or ingested. (Belpomme et al. 2007)	Lymphoma	Possibly HIV-related Kaposi’s sarcoma (WHO, 2006), increased relative risk - indoor Volatile Organic Compound exposure, Indoor use of insecticides (Irigaray et al. 2007)
Cervical	Human papilloma viruses (WHO, 2006)	Melanoma	Possibly UV exposure. (WHO, 2006)
Childhood leukemia	Extremely low-frequency electromagnetic fields (Boyd & Genius, 2008)	Non-Hodgkin’s lymphoma	Pesticides and triple the risk (Boyd & Genius, 2008)
Childhood Cancer	In-utero exposure to industrial chemicals, particularly those produced by fossil fuel combustion.	Kidney Cancer	Chromium or Nickel (Irigaray et al. 2007)
Leukemia	Indoor Volatile Organic Compound exposure, Indoor use of insecticides (Irigaray et al. 2007)	Skin Cancer	Asbestos in drinking water (WHO, 2006), environmental exposure to Arsenic and UV radiation. (Boyd & Genius, 2008)
Liver Cancer	Aflatoxins in food, Arsenic oxides if inhaled or ingested. (Belpomme et al. 2007)	Stomach Cancer	Helicobacter pylori, transmission may be facilitated by poor sanitation and crowding. (WHO, 2006)
Lung Cancer	Environmental tobacco smoke, radon, asbestos, chromium, nickel, cadmium, (WHO, 2006) environment exposure to arsenic, particulate air pollution, polycyclic aromatic hydrocarbons. (Boyd & Genius, 2008), Arsenic oxides if inhaled or ingested (Belpomme et al. 2007)	Prostate	Cadium (Irigaray et al. 2007)

respective etiological agents is provided below.

## Lung Cancer

*Environmental Tobacco Smoke (ETS):* ETS is associated with lung cancer and a number of studies support this relationship. The attributable fraction of lung cancer from ETS in those who never smoked and in those who were ex-smokers has been estimated to be 16-24% (Irigaray et al., 2007). A similar study among the residents in Western countries attributed 20-30% of lung cancer to ETS in those residents (Bukowski & Wartenberg, 1997). Boffetta (2002) assessed the incidence of lung cancer each year from ETS with an assumed Relative Risk (RR) of 1.2-1.3; this yielded 300 in Canada, 3000 in the United States and 1100 in the European Union (Boffetta, 2002).

*Outdoor Air Pollution:* The attributable fraction of lung cancer from traffic-related air pollution in those who never smoked and those who were ex-smokers was estimated to be 5-7%, as reported by Belpomme et al., 2007. Long term exposure to particulates and polycyclic aromatic hydrocarbons in adults is reported to increase the risk of lung cancer mortality by 8%, after controlling for tobacco smoke (Irigaray et al., 2007).

*Indoor Air Pollution:* A case control study in the Northern Province of South Africa, assessed lung cancer among women using wood or coal as main fuel at home. The increased risk of lung cancer was reported at 1.4 (95% CI 0.6–3.2) (Boffetta & Nyberg, 2003). Another study conducted in the Los Angeles area in 1981-1982 looked at cancer rates among white women that use coal for cooking and heating in the home during childhood and adolescence. This study reported an odds ratio of 2.3 (95% CI 1.0–5.5) for adenocarcinoma and 1.9 (95% CI 0.5–6.5) for squamous cell cancer (Boffetta & Nyberg, 2003).

*Radon:* Low radon levels in the home environment are the cause of approximately 10% of lung cancers (Belpomme et al., 2007). The estimated indoor radon exposure was estimated to be 59 Bq/m<sup>3</sup> which was determined by a population-weighted average of 29 studies. This radon exposure level would result in an attributable fraction of 4.5% for lung cancers (Boffetta, 2006).

The estimated lung cancer mortality in the United States due to radon and its decay products is 20,000 or more. This value corresponds to roughly 10% of all lung cancer

cases in the U.S. The average radon exposure to a U.S. single-family home is 40 Bq/m<sup>3</sup>, this could result in an excess lung cancer risk exceeding 1 in 1,000, with higher exposure levels causing a risk in excess of 1 in 100 (Bukowski & Wartenberg, 1997).

## Mesothelioma

*Asbestos:* Approximately 5% of the European population is believed to be exposed to residential asbestos according to the WHO. Although, according to Boffetta (2005) the studies selected for the meta-analysis were conducted with populations with high levels of exposure and a more reasonable estimation is in the order of 2% (Boffetta, 2005). A meta-analysis study estimated the RR of mesothelioma from environmental exposure to asbestos at 3.5 (95% CI 1.8–7.0). The corresponding RR to lung cancer was 1.1 (95% CI 0.9–1.5) (Boffetta & Nyberg, 2003). These results indicate that with a prevalence of exposure of 5% would lead to an incidence of 425 mesothelioma cases in men and 56 in women, and; a corresponding incidence of 771 lung cancers in men and 206 in women in the European Union (Boffetta & Nyberg, 2003).

A review and meta-analysis to assess the risk of pleura mesothelioma from environmental (household and neighbourhood) exposure to asbestos indicated an increased risk. The RRs of pleural mesothelioma for household exposure ranged from 4.0 to 23.7 and the summary risk estimate was 8.1 (95% CI 5.3-12) (Bourdès et al., 2000). For neighborhood exposure, RRs ranged between 5.1 and 9.3 (with a single RR of 0.2) and the summary estimate was 7.0 (95% CI 4.7-11). These results indicate an increased risk of pleura mesothelioma from high environmental asbestos exposure; although, the data was unable to offer the magnitude of the excess risk at levels which correspond to environmental exposure to the general population in industrial countries (Bourdès et al., 2000).

## Leukemia

*Magnetic Fields:* A study in England and Wales reported that those living within 200 metres from a high voltage power lines has a RR for leukemia of 1.69 (95% CI 1.13-2.52), and those who are born between 200 to 600 metres have a RR of 1.23 (95% CI 1.02-1.49) compared to those who lived >600 m from a line at birth. This study found a

significant dose-effect dependency in RR with relation to the distance from the line (Belpomme et al, 2007).

Belpomme et al, (2007) has estimated RR of 2 for acute leukemia for children living in areas with an average EMF strength above 0.4 mT and the EMF related RR for childhood leukemia is 2 for about 1% of the overall children population (Belpomme et al, 2007). However, Boffetta (2005) has reported that for childhood leukaemia the attributable fraction, based on the results of the pooled analysis, at 0.6%, corresponding to 18 cases per year in the European Union (Boffetta, 2005).

*Traffic pollutants:* Several researchers have found positive associations between local traffic density at the time of diagnosis and childhood leukemias. Children are mostly exposed to air pollutants through traffic exhaust; and the estimated RRs is between 1.6 and 4.7 (Belpomme et al., 2007).

## Bladder Cancer

A pooled analysis of six epidemiological studies have reported a summary RR of 1.18 (95% CI 1.06 - 1.32) for bladder cancer for exposures above 1 µg/L to trihalomethanes (Boffetta, 2006). However, caution must be used in interpreting these findings because people also consume water outside of their homes and from other sources, which is often overlooked in epidemiological studies. The concentration of chlorination by-products in water also varies greatly depending on geographical area, season and by organic contaminants. It is also necessary to consider confounders such as smoking, diet and other lifestyle related factors. It is believed that the environmental attributable fraction of bladder cancer is 10.3% which corresponds to, in the European Union, to 8911 cases in men and 2439 cases in women in 2002 (Boffetta, 2006). A review and meta-analysis of seven studies of bladder cancer risk from consumption of chlorinated water reported a RR of 1.21 (95% CI 1.09–1.34). This estimate was not modified after adjusting for smoking (Boffetta & Nyberg, 2003).

Other studies on bladder cancer in areas with low or intermediate contamination have shown an increased risk of bladder cancer from arsenic (Boffetta & Nyberg, 2003). An ecological study from Finland found a RR 2.44 (95% CI 0.95–1.96) with 3–9 years of latency, and 1.51 (95% CI 0.67–3.38) with 10 or more years of latency for exposure to arsenic at concentrations of higher than 0.5 µg/l compared

to less than 0.1 µg/l. In the United States, the RR for a dose of 53 mg or more of arsenic, as opposed to less than 19 mg of arsenic, and the RR was 1.14 (95% CI 0.7–2.9) overall, but the RR was 3.3 (95% CI 1.1–10.3) among smokers (Boffetta & Nyberg, 2003).

Ever consumption of chlorinated drinking water was associated with an increased risk of bladder cancer in men (combined OR=1.4, 95%CI 1.1-1.9) and women (combined OR=1.2, 95%CI 0.7-1.8). The combined OR for mid-term exposure in both genders was 1.1 (95% CI 1.0-1.2) and for long term exposure was 1.4 (95%CI 1.2-1.7). The combined estimate of the slope for a linear increase in risk was 1.13 (95% CI 1.08-1.20) for 20 years and 1.27 (95% CI 1.15-1.43) for 40 years of exposure in both sexes. This review and meta-analysis of the epidemiological literature indicates that long term consumption of chlorinated drinking water is associated with bladder cancer, particularly in men. (Villanueva et al., 2003).

## Brain Tumours

*Cell Phones:* There is inclusive evidence of increased risk of brain cancers from increased use of cell phones. About 25 epidemiological studies were identified that addressed cell phone use and brain tumours. Combined odds ratios (95% confidence intervals) from these studies for glioma, acoustic neuroma, and meningioma were 1.5 (1.2–1.8); 1.3 (0.95–1.9); and 1.1 (0.8–1.4), respectively. The overall evidence speaks in favor of an increased risk, but its magnitude cannot be assessed at present because of insufficient information on long-term use of cell phones. (Kundi, 2009).

## Skin Cancer

Existing data on the incidence of human skin cancer were analyzed, as available from two special surveys of non-melanoma skin cancer in the United States. The incidence of non-melanoma skin cancer in the ten regions that were surveyed not only correlated significantly with the ambient UV dose but also with the average daily maximum sunlight in summer. For squamous cell carcinoma the incidence was higher by 5.5% (SE 1.6%) per degree Celsius and for basal cell carcinoma by 2.9% (SE 1.4%) per degree Celsius. These values correspond to an increase of the effective UV dose by about 2% per degree Celsius. Although the precise nature of this correlation with temperature requires fur-

ther studies, it can be concluded that as the temperature rises the intensity of the sunlight increases and amplifies the induction of non-melanoma skin cancers by UV radiation in human populations. (Van der Leun et al., 2008).

## Conclusion

Although the term environment has been defined in different ways, this review has used the narrow definition proposed by the WHO. It is necessary to have consensus on appropriate definition for environment. Extensive research has been done to assess the etiological relationship between environmental agents and the risk of cancer. A number of studies have also quantified the risks for some of these agents. Lung cancer, mesothelioma, leukemia, bladder, brain and skin cancer appear to have identified environmental risk factors. Environmental tobacco smoke, indoor and outdoor pollution, radon, asbestos, magnetic fields, traffic pollution, trihalomethanes, cell phones and sunlight appear to be the most studied and reported. However, other environmental agents need to be identified and attributable fraction calculated.

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