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The environment carcinogen substance can contribute to cancer and health challenges: Impact action strategies

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Abstract
Cancer develops over several years and has many causes. Several factors both inside and outside the body contribute to the development of cancer. In this context, scientists refer to everything outside the body that interacts with humans as the "environment. Over the last 30 years scientists have worked hard to identify substances in the home, workplace, and general environment that cause cancer. This is a challenging task because there are more than 100,000 chemicals commonly used by Americans in household cleaners, solvents, pesticides, food additives, lawn care, and other products. Every year, another 1,000 or so are introduced. Furthermore, these are single substances and do not consider the mixtures and various combinations of commercial and consumer products that Americans are exposed to every day. In addition, many chemicals may be changed to different substances by the atmosphere, water, plants, and by incineration or combustion. Adding to the complexity, scientists know that cancer-causing substances are sometimes created during the synthesis or combustion of other chemicals. Dioxin is an example of this kind of unwanted contaminant effects on human health.

Keywords: Environment carcinogen, can contribute, health challenges

Introduction
Cancer refers to any one of many diseases characterized by the development of abnormal cells that divide uncontrollably and have the ability to infiltrate and destroy normal body tissue. Cancer often has the ability to spread throughout your body. Cancer is the second-leading cause of death in the world. But survival rates are improving for many types of cancer, In the United States, an estimated 15.5 million people with a history of cancer were living as of January 1, 2016, according to a 2018 report from the American Cancer Society.
In its broadest sense, the environment can be defined as external conditions influencing the development of people, animals or plants, and the purpose of studying the effects of the environment on human health, a distinction is often made between conditions from which individuals may have no or only partial control and those for which some element of personal choice exists. Exposure encountered at work and to substances in air and water would, for example, tend to fall in the former category, while "lifestyle" factors such as smoking, eating a high fat diet, and drinking alcohol would come in the latter. The environmental cancer is a term often limited to cancers resulting from chemical exposures, especially manmade, although most research workers use it in the wider sense to cover all conditions that impact on human cancer. This research gives an overview of environmental causes of cancer and the approaches used in the investigations of this issue, discusses the controversies and challenges, and outlines some of the emerging scientific methodology.
Environment Influence: Many different sources, processes, and pathways can lead to exposure to an environmental hazard and adapted and shows some of the elements involved in the process from source to heath effect. Humans are exposed to a wide range of potential natural or synthetic toxicants, and frequently to a multitude of complex mixtures. Many studies have a tendency to focus on a limited range of environmental influences, often because of the restricted nature of the group under investigation for example, in specific employment sectors or because of difficulties in obtaining information on the wider environment. A more holistic approach is needed, however, with consideration of estimates of total body burden to potential carcinogenic substances, in order to set specific environmental influences in context and to inform the development of effective risk

Reduction strategies. Contamination of food by environmental chemicals such as dioxins, polychlorinated biphenyls (PCBs), and pesticide residues is of considerable public concern. It has been established that the food chain is the primary pathway of human exposure to dioxins and PCBs, particularly from meat, dairy products, and fish. The widespread exposure to these substances can be exemplified from the fact that they can be detected in most human adipose tissue. They are also present in breast milk and, on a body weight basis, the intake of breast-fed babies has been estimated to be one or two orders of magnitude higher than the average adult intake. Internal Agency on Cancer (IARC) has classified dioxin as a human carcinogen based on limited evidence in humans and sufficient evidence in experimental animals as well as on mechanistic considerations.

A large range of pesticides is widely used in the production of food and the potential harmful effects from pesticide residues in foodstuffs continue to raise public concern. Current regulation of pesticides should ensure that substances that are shown to be carcinogenic do not reach or are withdrawn from the market. However, compounds such as some chlorinated organic pesticides (for example, DDT) were used extensively in the 1950s and 1960s. These organochlorine compounds have long persistence in the environment and in animal tissues. The presence of metabolites in human tissue has been suggested to be an etiological factor in the development of some cancers for example, breast cancer.

In this article, studies the types of cancer, how the disease develops, and the many treatments that help improve the quality of life and survival rates. Each year, more than 40,000 people in the country receive a diagnosis of one of the following types of cancer:

- bladder
- colon and rectal
- endometrial
- kidney
- leukemia
- liver
- melanoma
- non-Hodgkin's lymphoma
- pancreatic
- thyroid

Other forms are less common. According to the National Cancer Institute,

Human cancer is really not a single disease but is a family of different diseases. The following factors were selected that appear to increase the risk of different types of human cancers:

Table 1: Examples of biological, biophysical and biochemical association with human cancers

Carcinogens	Cancer Sites	Occupational Sources
Arsenic	Lungs, Skin	Electricians, Smelters, Medications.
Asbestos	Mesothelioma, Lungs	Roof and floor tiles
Benzene	Blood and lymph nodes	Petroleum, painting, detergent, rubber
Beryllium	Lungs	Missile fuel, Nuclear reactor
Cadmium	Prostate	Battery, painting and coating
Chromium	Lung	Preservatives, pigments, paints
Ethylene oxide	Blood	Ripening agent for fruits, gases
Nickel	Nose, Lungs	Battery, Ceramics, Ferrous alloys
Radon	Lung	Uranium decay, Mines, Cellars
Vinyl chloride	Liver	Refrigerator, glues
Smoke	Lungs, Colon	Cigar, air pollution, car smoke
Gasoline	Lung, Blood	Oil petroleum
Formaldehyde	Nose, Pharynx	Hospital/laboratory workers
Hair dyes	Bladder	Hairdresser and barber
Soot	Skin	Chimney cleaners
Ionizing radiation	Bone marrow	Radiology technician
Hepatic virus- B,C	Liver	Hospital workers, drug users
HPV/Herpes viruses	Cervix, skin, head/neck	Multiple sexual partners
Burkitt’s virus	Lymph node	Black people in South Africa
Helicon-bacteria pylori	Stomach	Chronic bacterium infection

Environmental Pollution

The proportion of cancers attributed to environmental pollution is even smaller than that for occupation.

Ambient air pollution:

There have been many studies of cancer and air pollution, particularly ambient air (reviewed by Rushton For example, urban air contains thousands of chemicals and Cohen and Pope suggest that lung cancer, in particular, may be increased by ambient air pollution, chiefly due to the incomplete combustion of fossil fuels. A considerable number of studies have compared lung cancer rates in urban and rural residents of the same country, or lung cancer rates for urban areas stratified according to population size and have showed increased incidence in urban areas. This relationship has been further explored in a series of studies of changes in lung cancer experience of immigrants to various countries. For example, several investigations on emigrants from the UK to New Zealand showed that, in general, the lung cancer rates of emigrants were lower than that of residents in the UK but higher than those born in the new country. It has been suggested that this reflects a lasting effect of early environment on lung cancer mortality later in life.

Some environmental studies have indicated that lung cancer is more closely related to sulfates as an index of air pollution than fine particles and this is supported by studies of occupational groups and animal experiments. Other constituents of air that have been associated with increased lung cancer include asbestos, polycyclic hydrocarbons, and diesel exhaust. The evaluation of the role of air pollution in

the etiology of other cancers is even more equivocal than for lung cancer. Associations have been suggested, for example, with digestive and gastrointestinal tract cancers, bladder cancer, esophageal cancer, and breast cancer.

Indoor Air

In addition to ambient air pollution, the potential contribution of indoor air pollution, particularly environmental tobacco smoke to the risk of lung cancer, has been increasingly recognized as important, given that, in developed countries, many people spend up to 90% of their time indoors. Levels of substances in indoor air are related to levels outdoors, the activities of the occupants (for example, smoking, cooking), fitting and furnishings (for example, formaldehyde in insulating materials), and the geographical location (for example, radon). The contribution of environmental tobacco smoke and radon on the incidence of lung cancer has been estimated to be considerable, with approximately 2% of lung cancer deaths occurring in non-smokers. The source of formaldehyde, classed by IARC as a group 2A probable human carcinogen, in indoor air is mainly from furnishing materials and cigarette smoke. Levels indoors have been shown to be 10 times greater than levels outdoors.

Water

There are numerous studies relating to water pollution, especially the byproducts of chlorination, although the evidence that water pollution is an important factor in human cancer is unconvincing. Potential contaminants of concern include arsenic, which at high doses has been shown to cause skin cancer when ingested. The byproducts of chlorination of water-primarily halogenated organic compounds, including trihalomethanes such as chloroform-have been associated with an increased risk of both bladder and rectal cancers, although the influence of diet was not explored in these studies.

Radiation

Exposure to all forms of radiation, including ionizing radiation, UV light, and low frequency sources, causes public concern. Doll suggests that ionizing radiation may cause up to 4% of all cancers, mostly as a result of natural radiation from radon and cosmic rays, external radiation from radionuclides in rocks, soils, and building materials, and internal radiation from naturally radioactive traces in food. Radon concentrations indoors vary greatly depending on local geological characteristics. Estimates of annual cancer deaths caused by indoor radon exposure also vary widely. Doll suggests that up to 6% of lung cancer in the general populations may be due to radon and that action to reduce the risk is advisable.

UV light from sunlight exposure is responsible for a large number of skin cancers, including melanomas and basal cell and squamous carcinomas. Squamous carcinomas appear to be related to cumulative exposure to UV light while melanoma, which has been steadily increasing in all white skinned populations for many years, is associated with frequency of sunburn. There is also evidence to suggest a relation between UV light and the risk of non-Hodgkin's lymphoma and chronic lymphatic leukemias, with squamous cell carcinomas and melanomas being found to be much more common than would be expected after the occurrence of non-Hodgkin's lymphoma and chronic lymphatic leukemia. Doll suggests that, if the relation is confirmed in other studies, it might be explained by the effect of UV light on the immune system, strengthening even more the need to avoid unnecessary exposure to UV light.

Electromagnetic fields experienced through living near cables, radio and telegraph operations, and other sources have been a topic of increasing public concern, particularly in relation to emissions from mobile phones and from the base stations that receive and transmit the signals. An expert group which examined the possible effects of these sources of radiation concluded that exposures of the general population from base stations emissions and other emitting sources such as antennae for radio, television, and paging are well below guideline values. The levels of exposure arising from phones held near the head are, however, substantially greater than these.

Cognitive tests on volunteers have found that mobile phone signals shorten reaction times in some tasks. It has been suggested that these reflect the effect of small temperature increases on synaptic transmission in the region of cerebral cortex directly under the headset antenna. To date, few epidemiological studies have directly examined the relation of mobile phones to morbidity or mortality, and none has explored the effects from base stations, although there have been several ecological studies of this aspect. Overall the evidence to date does not indicate that risks are increased, although many studies lack statistical power, and some have methodological deficiencies. The expert group recommended a range of issues that needed further urgent research.

Waste management activities

The vast amount of household and commercial waste, together with smaller quantities of industrial and specialized wastes, including that from hospitals, is disposed of mainly through incineration or in landfill sites. The potential health effects of substances emanating from both these sources have been the subject of many studies. Incineration can give rise to a wide range of gases and aerosols, including fine particulate matter and many metals and organic chemicals, many of which have potential toxic properties. Highest public concern has been raised about dioxins, PCBs, and polycyclic aromatic hydrocarbons (PAHs). A report by the Institute for Environment and Health reviewed the 10 pollutants most likely to be produced in amounts sufficient to exceed air quality standards or to be of concern because of their toxicity. Established or potential carcinogenic compounds included cadmium, arsenic, chromium, nickel, dioxins, PAHs, and PCBs. Although many of these compounds have been shown to be carcinogenic in occupational studies with high levels of exposure, the evidence of cancer risk at the much lower levels resulting from incineration is either lacking or equivocal.

The health of populations living near waste landfill sites has been reported in a number of ecological studies. A general limitation of many of these studies is the imprecise information on exposures from the sites through the use of surrogate measures such as distance from a site. Very few collect data on specific substances or confounding factors. The evidence for a causal relation between landfill exposures and cancers is weak, although several studies have reported excesses of bladder, lung, and stomach cancer and leukemia.

Controversies and Challenges

In a report of the President's Cancer Panel conference on avoidable causes of cancer held in 1994, Davis and Muir suggest that studying the effects of environmental pollution remains one of the most challenging areas of epidemiologic research involving large numbers of people whose exposures change over time and are often poorly characterized. A lack of exposure data is the most commonly cited factor preventing identification of a causal association between environmental and occupational risk factors and advanced health effects.

Whatever the type of study design, the results are ultimately dependent on the quality of the data. As Higginson points out, global cancer statistics of good quality have now become available. The identification of populations exposed to environmental contaminants may be difficult because of inadequate identification of specific carcinogens and the distribution of contaminants in the environment, and knowledge of the duration and concentration of specific carcinogens over many years of exposure. It is also important to be able to identify sensitive population subgroups-that is, those who are particularly susceptible to the effects of a pollutant-and to be able to evaluate the variation of both individual susceptibility and individual dose.

The increasing awareness of the potential health hazards and environmental impacts of the possible pollution of the environment by man-made chemicals has led to the development of "cancerphobia" and the tendency to blame the environment for cancer occurrence. In a series of papers (for example, Ames and Gold, and Ames *et al.* Ames suggests that the idea that traces of synthetic chemicals are major contributors to human cancer is not supported by the evidence. After adjustment for age, and excluding lung cancer, cancer death rates in many countries are in fact showing decreasing trends. Ames emphasizes the need to put the possible hazards of man-made carcinogens into perspective and points out that there is an enormous background of natural chemicals (roasted coffee, for example, contains more than 1000), many of which have been shown to be rodent carcinogens.

Ames and Gold suggest that the factors which are likely to have a major effect on reducing rates of cancer include:

- reduction of smoking
- increased consumption of fruits and vegetables
- control of infections
- avoidance of intense sun exposure
- increased physical activity
- Reduced consumption of alcohol and possibly red meat.

They feel that it is important not to divert society's attention away from the few really serious hazards by the pursuit of hundreds of minor or non-existent hazards.

Davis and Muir advocate the use of the "precautionary principle"—that is, society should take care not to engage in activities which appear likely to increase risks, even though uncertainty exists about the size and extent of those risks. They suggest that even though the cellular and genetic mechanisms of cancer are not fully understood, basic improvements in lifestyle, such as diet, smoking (and other drug) habits, and exercise, and in our chemical–physical environment, such as reduced toxic emissions, might have a beneficial effect on general public health, in a similar way to the improvements observed in infectious diseases occurrence in the 19th century. Multiple exposures or combinations of low levels of commonly occurring carcinogens could be part of the explanation for persisting patterns of cancer that are otherwise inexplicable.

Two divergent cancer control strategies have emerged. The first, supported by environmental activists and regulators, is to build regulatory programs to control or eliminate pollutants in the ambient environment. The second is to direct effort into understanding the fundamental biological mechanisms of carcinogenesis, with the aim of intervening in this process through chemoprevention or treatment.

The first approach has been successfully implemented in the occupational area, where the gradual introduction of standards and exposure units has resulted in reduced levels of hazardous substances. Environmental regulation of ambient air and water has also resulted in decreasing levels of polluting substances. Logically, primary prevention of occupational and environmental carcinogens should result in lower cancer rates. The challenges facing researchers to develop appropriate methodology and carry out suitable studies to quantify changes include:

- the investigation of the relative roles of different sources of a potential carcinogen—that is, total exposure estimation
- development of epidemiological methodology to detect effects of very low levels and simultaneously evaluate the impact of potential confounding factors
- extension of knowledge of the molecular pathways and corresponding precursors of different cancers
- Identification of sensitive subpopulations and evaluation of the role of individual susceptibility.

Peto, suggests that the rapid advances in genetic and molecular understanding will increasingly facilitate the quantification of relations between risk factors and specific events in carcinogenesis. The field of molecular epidemiology offers the opportunity to combine the scientific disciplines of epidemiology and molecular toxicology to investigate the interactions between genetic factors and environmental factors in the cause of disease. In long term studies of occupational cohorts and wider populations it has been shown that exposures to relatively high levels of established carcinogens over long periods do not affect all individuals equally. For example, only a fraction of the population of heavy smokers develops lung cancer. There is increasing evidence that genetic factors may influence an individual's susceptibility and resistance to cancer.

There is substantial inter-individual variation in genes whose products metabolize carcinogens and anti-carcinogens, repair DNA damage, and maintain cell cycle control and immune function. Since Doll and Peto published their estimates of the different causes of cancer there has been increasing research into the multiple pathways involved in the carcinogenic process and the importance of interactions. These include simultaneous exposure to both different causal and different protective factors, and host–environment (endogenous versus exogenous) interactions, including metabolic polymorphisms. Many of the genes that encode the enzymes that metabolize potential carcinogens are polymorphic-that is, there are common variant forms (prevalent in at least 1–2% of the population), resulting from genetic mutations passed down through generations. However, it should be noted that some genes may encode an

enzyme that can detoxify a potential carcinogen and act on another non-genotoxic substance to produce a carcinogen.

The investigation of genetic susceptibility can range from the identification of "high penetrance" mutations, which are rare in the general population but for which the lifetime cumulative risk of the development of cancer is very high, to "low penetrance" mutations which are relatively common but have a low relative risk. The latter require interaction with environmental risk factors and, because they are common, the fraction of disease caused by a particular polymorphism (that is, the attributable risk) may be substantial and thus have important public health implications. An example of a high penetrance mutation is the BRCA1 gene that gives a high risk of breast cancer to the small proportion of women who are carriers. An example of the result of low penetrance mutation is the slow acetylator version of N-acetyltransferase 2 (NAT2) that has been shown to increase the risk of bladder cancer in people exposed to amylamines. However, the fast acetylator version which "protects" from bladder cancer may also increase the risk of colon cancer.

Future research will need to incorporate measurement of susceptibility to aid the investigation of carcinogenic pathways and to detect gene–environment interactions. This will require multidisciplinary collaborative teams involving epidemiology, toxicology, and exposure assessment. Numbers of scientific and media reports of cancer studies have risen dramatically in the last decade. Perhaps the greatest challenge for the research community will be the requirement to communicate clearly key concepts and principles of interpretation, including the difference between a statistical association and a causal association with biological meaning. The limitations and uncertainties of a study and its contribution to existing scientific evidence will facilitate the ongoing debate of identifying and correcting misperceptions about cancer risk factors. Above all, as Linet says, it will be important for researchers to listen more closely to the concerns expressed by members of the public, the media, and policy makers.

Community Impact Action Strategies: There are multiple ways that public and private organizations at the local, state, and national levels can develop policies and allocate or expand resources to facilitate necessary changes. Schools can ensure that students participate in physical activity programs and promote the availability of healthful food and beverages while reducing access to less healthy foods and beverages. Employers can implement worksite health promotion programs. Health care professionals can advise and assist their patients on effective weight loss and weight management programs. At the state and local level, community leaders, in particular, can promote policy changes that may include regulation of the school food environment, zoning changes, tax incentives that bring food stores that carry fresh fruits and vegetables into poor neighborhoods, and the creation of safe spaces that promote physical activity. A growing number and variety of policies are being implemented at the local and state levels of government that are intended to promote healthy eating and active living; many of these policies have targeted the food environment and activity requirements in schools. These varied efforts can become an extremely valuable source of useful information about the impact of different policy strategies to the public's response in terms of consumption of certain products or influence on dietary behaviors and weight management. The Centers for Disease Control and Prevention (CDC), the Institute of Medicine, the World Health Organization, and others have outlined a variety of evidenced-based approaches in schools, worksites, and communities to halt and ultimately reverse obesity trends. Some specific approaches are outlined in support evidence-based cancer prevention strategies that reduce barriers to healthy living through research, education, outreach, health promotion programs, and advocacy. We are actively collaborating with others to advance some of these strategies in states and localities nationwide.

Conclusion

Cancer is a complex genetic disease as a consequence of environmental exposures which serve as the driving force in initiating tumor development and progression. The scientific literatures provide substantial evidences of environmental and occupational causes of cancer. This will fully support an accelerated effort to prevent carcinogenic exposures. In addition to all of the evidences cited, there are many other indications that environmental exposures are linked to various human cancers. The single major risk factor for cancer is age, and the number of our geriatric people is rapidly increasing. If we look only at incident patterns among those aged 65 and 85 years old, there will be a significant increase number of cancer patients over the past 30 years. The same is correct for other ages as well. Cancer has become a widespread disease with epidemic proportions in certain cancer sites in a single generation. Currently, about one in four Americans could expect a cancer diagnosis at some point during his or her lifetime.

People can avoid some cancer-causing exposures, such as tobacco smoke and the sun's rays. But other ones are harder to avoid, especially if they are in the air we breathe, the water we drink, the food we eat, or the materials we use to do our jobs. Scientists are studying which exposures may cause or contribute to the development of cancer. Understanding which exposures are harmful, and where they are found, may help people to avoid them. But others are the result of environmental exposures that damage DNA. These exposures may include substances, such as the chemicals in tobacco smoke, or radiation, such as ultraviolet rays from the sun.

The United States has regulations in place to limit exposure to carcinogens in the workplace and elsewhere in the environment. The National Toxicology Program and the International Agency for Research on Cancer publish lists of known or suspected cancer-causing substances. Individuals can reference these lists to understand the risks associated with contact with certain carcinogens and limit their exposure.

From the womb to old age, people around the world are exposed to countless carcinogens in their food, air, water and consumer goods.

The National Institutes of Health has classified 54 compounds as known human carcinogens based on studies indicating they cause at least one type of cancer in people, according to the nation's 11th Report on Carcinogens. The highest exposures occur in an occupational setting, but there are environmental exposures as well.For example, benzene, a known cause of human leukemia, is a common pollutant in vehicle exhaust. Radon, a natural radioactive gas found in many homes, raises the risk of lung cancer. Arsenic, linked to skin, liver, bladder and lung cancer, contaminates some

drinking water supplies. Other known human carcinogens include asbestos, hexavalent chromium, aflatoxins and vinyl chloride. Since 1981, agencies and institutes have cited the same estimate when regulating carcinogens in the workplace, air, water and consumer products. Roughly four percent of cancer deaths – or 20,000 deaths per year - may be attributable to occupational exposures, and two percent – or 10,000 deaths per year – to environmental exposures. In its new report, the panel, appointed by former President Bush, called that estimate "woefully out of date," reporting that "the true burden of environmentally induced cancers has been grossly underestimated."

In its broadest sense, the environment can be defined as external conditions influencing the development of people, animals or plants. For the purpose of studying the effects of the environment on human health, a distinction is often made between conditions from which individuals may have no or only partial control and those for which some element of personal choice exists. Exposure encountered at work and to substances in air and water would, for example, tend to fall in the former category, while "lifestyle" factors such as smoking, eating a high fat diet, and drinking alcohol would come in the latter. Higginson[1] points out that, in the public mind "environmental cancer" is a term often limited to cancers resulting from chemical exposures, especially manmade, although most research workers use it in the wider sense to cover all conditions that impact on human cancer.

This paper gives an overview of environmental causes of cancer and the approaches used in the investigations of this issue, discusses the controversies and challenges, and outlines some of the emerging scientific methodology.

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