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Short Communication

Exposure to Carcinogens and its Effects in the Formation of Cancer

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ABSTRACT

A carcinogen is any substance which can arise in both natural and synthetic substances, radionuclide or radiation that promotes carcinogenesis and formation of cancer. This is due to the ability to damage the genome or to the disruption of cellular metabolic processes. Radioactive substances are considered as carcinogens, but their carcinogenic activity is related to the radiation, example gamma rays and alpha particles. Some examples of non-radioactive carcinogens are inhaled asbestos, certain dioxins and tobacco smoke. Carcinogens are not immediately toxic thus their effect can be in a gradual way.

INTRODUCTION

Cancer is a disease in which normal cells are damaged and do not undergo programmed cell death as fast as they divide via mitosis. Carcinogens increase the risk of cancer by changes in cellular metabolism or damaging DNA directly in cells and it also interfere with biological processes and induce the uncontrolled malignant division leading to the formation oftumors. Usually, severe DNA damage leads to programmed cell death, but if the programmed cell death pathway is damaged, then the cell cannot prevent itself from becoming a cancer cell [1].

There are many natural carcinogens. Aflatoxin B1, produced by the fungus Aspergillusflavus is an example of naturally occurring microbial carcinogen. Some viruses such as hepatitis B and human papilloma virus, found to cause cancer in humans. Other infectious organisms which cause cancer in humans include some bacteria (e.g. Helicobacterpylori) and helminths (e.g. Opisthorchisviverrini and Clonorchissinensis).

Dioxins and dioxin-like compounds, benzene, kepone, EDB, and asbestos are considered as carcinogenic. Industrial smoke and tobacco smoke were sources of carcinogens. Benzo[a]pyrene, tobacco-specific nitrosamines such as nitrosonornicotine, and reactive aldehydes such as formaldehyde is also a hazardous. Vinyl chloride, from which PVC is manufactured, is a carcinogen and thus a hazard in PVC production [1].

COMMENTARY

The link between tobacco products and human cancers results from a nicotine and carcinogens. Without either one of these, tobacco is the single greatest cause of death due to preventable cancer. Nicotine is addictive and toxic, but it is not carcinogenic. This addiction causes people to use tobacco products continually, and these products contain many carcinogens [2].

People are continuously exposed exogenously and endogenously to varying amounts of chemicals that have been shown to have carcinogenic or mutagenic properties. Exposure can occur exogenously through carcinogens present in food, air or water, and endogenously when carcinogens are present in products of metabolism or pathophysiologic states such as inflammation. Exposure to environmental chemical carcinogens may contribute a majority of human cancers, when exposures are related to "life-style" factors such as diet, tobacco etc. Factors also include Carcinogens in tobacco products, carcinogenicity in laboratory animals, human uptake, metabolism and adduct formation, possible role in causing molecular changes in oncogenes or suppressor genes, and other relevant data.This approach can be applied to evaluation of other environmental carcinogens, and the evaluations would be markedly facilitated by prospective epidemiologic studies incorporating phenotypic carcinogen-specific biomarkers [3].

Janus carcinogens are carcinogenic agents under differing conditions of cell type or dose also acts as anticarcinogens. The carcinogenicity and anticarcinogenicity of tobacco smoke and/or tobacco itself is due to components in the mixture, not that of a

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single carcinogenic chemical that also may be anticarcinogenic [4].

The transformation of chemicals is important in carcinogenesis, both in bioactivation and detoxification. Pathways of transformation include nitropolycyclic hydrocarbons, polycyclic hydrocarbons and their diols, vinyl halides and dihaloalkanes. Advances in analytical methods and recombinant DNA technology contributed greatly to the study of metabolism of chemical carcinogens [5].

Mutational mechanisms contribute to the activity of most human carcinogens. Many of these chemicals are electrophilic or are metabolically activated to reactive molecules that can alter DNA. Human carcinogens that do not exhibit direct chemical interaction with DNA such as hormones and asbestos shown to induce genetic effects when in vitro assays for chromosomal mutations [6].

Per- and polyfluoroalkyl substances (PFAS) is a large class of environmentally persistent chemicals used in industrial and consumer products. Human exposure to PFAS is extensive, and PFAS contamination has been reported in drinking water and food supplies as well as in the serum of nearly all people. Perfluorooctanoic acid (PFOA), member of the PFAS class, induces tumors in animal bioassays and associated with elevated risk of cancer in human populations. GenX, one of the PFOA replacement chemicals, induces tumors in animal bioassays as well [7].

The environmental burden of cancer fall between previously estimated burdens of alcohol and tobacco use. The results allow for a comparative assessment across carcinogens and offer insights into strategies to reduce the environmental burden of cancer [8].

Not all carcinogens are mutagens, and many mutagens are not carcinogens. Among related chemicals, small changes of structure can also influence carcinogenic potency. Many tumours are genetically unstable. Benign types rarely exhibit 'progression' or show evidence of genetic instability. Cells of particular tumour types exhibit identifiable particular 'sets' of phenotypic abnormalities. Tumour cells pass their abnormalities on to their daughter cells, indicating that a genomic alteration probably underlies tumour formation [9].

Carcinogens present in tobacco products, the mechanisms by which tobacco causes cancer, and the various tumor types causally related to tobacco use [10]. Consumption of white meat, such as pork and poultry meat are positively or inversely associated with an increased risk other types of cancer [11].

CONCLUSION

This commentary shows how different types of carcinogens may increase the risk of cancer by altering cellular metabolism or damaging DNA directly in cells, which interferes with biological processes, and induces the uncontrolled, malignant division, ultimately leading to the formation of tumors.

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