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Editorial

Mechanism of Cancer

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INTRODUCTION

In the past decade, we are gaining more insight of cancer mechanism. The association between environmental factors such as diet, irradiation, infection, and cancer have long been recognized through epidemiology studies. Host susceptibility to cancer has been increasingly recognized through the studies of familial cancer syndromes, single nucleotide polymorphism etc. Carcinogenesis probably results from the interaction between environmental factors and host susceptibility, and is a multi-steps process. Involving a number of somatic genetic alterations through a series of morphological changes. These include activation of oncogenes, silencing of tumor suppressor genes through mutation, loss of heterozygosity or CpG island methylation. The epigenetic change, CpG island methylation, is increasingly being recognized to be an early and important mechanism for tumor suppressor gene silencing. Epigenetic silencing does not involve the changes of nucleotide sequence. Hence it is potentially reversible and may potentially offer early chemoprevention. These mechanisms are discussed in detail in this review.

ENVIRONMENTAL CAUSES

Cigarette smoking, as one of the most important environmental causes for cancer, is the direct and avoidable cause of an enormous cancer burden. No other known single environmental factor has anything like the same degree of importance for cancer in the developed world.

Radiation carcinogenesis is a twentieth century problem. One of the most important pieces of information comes from the long-term follow-up of survivors of the atomic bombs dropped in Nagasaki and Hiroshima. Studies of occupational exposure of diagnostic radiologists, uranium miners, and workers in the nuclear industries have also provided valuable information. More has come from the analysis of cancer incidence in patients exposed to radiation for medical purposes, either for diagnosis or for treatment of non-malignant conditions.

UNIFYING THEMES IN CARCINOGENESIS

Only 5-10% of cancers are hereditary. Most cancers develop through genetic alterations due to damage by environmental factors at a background of host susceptibility. These genetic changes are called acquired/somatic genetic alterations, which usually take many years before they develop into a cancerous cell.

Uncontrolled cell growth and metastasis. This loss of normal control mechanisms arises from the acquisition of alterations in three broad categories of genes:

(1) Proto-oncogenes, the normal products of which are components of signalling pathways that regulate proliferation and which, in their mutated form, become dominant oncogenes;

(2) Tumor suppressor genes, which generally exhibit recessive behaviour, the loss of function of which in cancers leads to deregulated control of cell cycle progression, cellular adhesion etc., and

(3) DNA repair enzymes, which promote genetic instability when mutated or epigenetically silenced

METHYLATION AND CARCINOGENESIS

Altered methylation patterns are known to occur in the DNA of cancer cells. Two ways have been observed: wide areas of global hypomethylation along the genome, and localized areas of hypermethylation at certain specific sites, the CpG islands, within the gene promoter regions.

DNA METHYLATION AND MUTATION

DNA methylation can predispose to mutations through cytosine deamination, which converts cytosine directly to thyrnine while the unmethylated cytosine is converted to uracil, which is recognized and repaired more efficiently. The increase mutability of 5-methylcytosine versus cytosine is influenced by three factors: differential repair efficiency, rate of spontaneous deamination, and rate of cell division. The mutagenicity of methylated cytosine in biological systems was first demonstrated by the

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observation that sites of cytosine methylation corresponded to mutational hotspots in the Lad repressor gene of E. Coli.

CONCLUSION

We are gaining more insight and understanding to the underlying mechanism for carcinogenesis. Early detection or screening is now possible for a number of cancers. We shall aim at primary cancer prevention by leading a healthy life style.