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Short Communication

A Short Note on Epigenetics Changes in Cancer

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DESCRIPTION

Changes in genes that control cell proliferation, survival, and other homeostatic processes, such as oncogenes and tumour suppressors, cause cancer. Genes are altered in cancer cells either by mutations, which change the function of the proteins they encode, or by epigenetics, which involves chromosome alterations that change gene expression patterns. DNA methylation, as well as the methylation, acetylation, or phosphorylation of histones and other proteins that wrap DNA to create chromatin, can cause this. Little is known about how these chemical alterations affect the expression patterns of oncogenes and tumour suppressor genes in cancer cells' DNA, although they have the potential to do so. DNA methylation, for example, causes "epigenetic silence," or the loss of tumour suppressor gene expression, which causes normal cells to die. Although little is understood about how these chemical alterations arise in cancer cells' DNA, they can affect oncogene or tumour suppressor gene expression patterns. DNA methylation, for example, causes "epigenetic silence," or the lack of expression of tumour suppressor genes, resulting in the transformation of normal cells into cancer cells [1-3].

The American Association for Cancer Research recently held a conference on Chromatin, Chromosomes, and Cancer Epigenetics in Waikoloa, Hawaii, which drew nearly 300 of the world's top researchers in this field. They reviewed the most prevalent chromosome areas and genes that undergo epigenetic alterations in cancer cells, as well as methods for detecting epigenetic modifications in cancer cells and potential therapeutics for reversing this process.

Mechanisms of epigenetic gene silencing

Stephen Baylin of Johns Hopkins University in Baltimore, Maryland, gave an overview of the basic principles behind epigenetic gene silencing in his inaugural presentation. Hyper methylation of genes' promoter regions, which are untranscribed portions of the genes that turn on and off transcription, can mute them. This methylation is most common in DNA sequences known as "CpG islands," which are usually found near gene promoters but can also be found elsewhere on a chromosome [4].

Hypermethylation has been found in the promoter regions of genes that control processes like proliferation, apoptosis, DNA repair, and immortalization in cancer cells. As a result, silencing genes that control these processes can encourage tumour creation and progression. In mice, for example, methylation silencing of SFRP genes causes cell proliferation and the creation of early dysplastic colon mucosal lesions because their products oppose the WNT signaling pathway. The cyclin-dependent kinase inhibitor p16Ink4 gene which is a tumour suppressor is methylated, which causes immortalization of breast and lung epithelial cells, which is one of the first steps toward becoming a cancer cell. In the majority of prostate tumours, the gene encoding the detoxifying enzyme glutathione S-transferase (GSTP1) is hypermethylated and inactive [5].

DNA compaction into chromatin is mediated by histones and other proteins. Histone acetyltransferases acetylate histones at specific amino acids, causing chromatin to take on a relaxed, or open, shape (also known as "euchromatin"), allowing transcription factors to access the DNA and genes to be expressed. Histone deacetylases (HDACs) deacetylate histones, causing chromatin to shut (also known as "heterochromatin"), preventing transcription factor access and gene activation. Changes in the activity level of HDACs, like DNMTs, can affect gene transcription that regulates cell-cycle progression and developmental events.

As a result, a number of inhibitors for DNA methylation and histone deacetylation in cancer cells have been produced [6]. The azanucleosides 5-azacytidine and 5-aza-2'-deoxycytidine (decitabine), which are the most clinically advanced drugs, were found more than 25 years ago when their methylation-inhibitory effects, even at low doses, were characterized for the first time. Despite the fact that both of these medicines were originally used at high dosages, their redesign for low-dose regimes has proven therapeutic activity. 5-Azacytidine has been demonstrated to be effective in patients with acute myeloid leukaemia and is currently licenced for the treatment of myelodysplastic syndromes (AML).

CONCLUSION

Epigenetic alterations are useful not only as therapeutic targets, but also in determining patient prognosis and predicting response to therapy. Methylation of the O6-methylguanine-DNA methyltransferase (MGMT) gene, which encodes a DNA-repair enzyme, inhibits the killing of tumor cells by alkylating agents. Studies have shown that methylation of the MGMT promoter in patient glioma samples is a useful predictor of the responsiveness of the tumors to alkylating agents. When this gene is silenced by methylation, patients are likely to have a better outcome after therapy. The effects of gene silencing on tumor formation and growth can therefore be both positive and negative.

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