

# Epigenetics - Role as Biomarker in Cancer Diagnosis

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Epigenetics alludes to change in the gene expression levels without bringing any alteration in DNA sequence. DNA methylation and Histone modifications are considered as significant epigenetic mechanisms that confer the heritable changes in cellular phenotype. These play a vital role in DNA based processes like Replication, Transcription and DNA repair. Consequently, genomic alterations or abnormal expression in chromatic regulators can have profound effect leading to induction of Cancer. Hypermethylation of CpG islands located in Promoter regions of tumor suppressor genes is considered to be important mechanism for gene inactivation. Hypomethylation refers to the reduced levels of global DNA methylation which promotes the different types of malignancies leading to cancer. Histone acetylation involves the regulation of chromatin structure leading to the increased or decreased levels of gene transcription. HAT and HDAC are the enzymes involved in the addition and removal of acetyl groups from lysine residues on the histone N-terminal tails. Histone methylation is carried out by conserved proteins known as HMTs which facilitates the addition of methyl groups to the amino terminals of histone proteins and is related to different biological processes ranging from transcriptional regulation to epigenetic silencing. DNA methylation is linked with many key processes like telomeres, centromeres, X-chromosome inactivation, and suppression of repetitive elements, genomic imprinting and carcinogenesis. There are two types of abnormal DNA methylation associated with human malignancies. Global hypomethylation is often associated with chromosomal instability and loss of imprinting whereas hypermethylation occurs at CpG islands located in Promoter regions and often associated with inactivation of tumor suppressor genes.

Epigenetic aberrations have an impact on the stages of tumorigenesis, eventually promoting the neoplastic cells with increase in pathogenicity. Identification of those alterations can be used as prognostic biomarkers for diagnosing Cancer at the early onset of the disease. These biomarkers will be helpful for characteristic patients whose malignancies are sensitive to particular cytotoxic chemotherapies that can hold guarantee for anticipating from which patients will be benefited from newer agents targeted at oncogenes. These biomarkers will be useful for trademark patients whose malignancies are delicate to particular cytotoxic chemotherapies that can hold guarantee for anticipating from which patients will be profit by more current focused on operators coordinated at oncogenes.

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