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Cell & Developmental Biology

Perspective

Determination of DNA Methylation by Bisulfite Modification

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DESCRIPTION

Heritable structural and biochemical modifications of chromatin without modifying DNA sequence are referred to as epigenetics. Epigenetic mechanisms control a variety of physiological and pathological processes by regulating relevant gene expressions and altering the accessibility of epigenetic codes to chromatin on a local and global scale. DNA methylation, histone modifications, and non-coding RNAs (ncRNAs) are the three main epigenetic codes that have been extensively researched. The most important epigenetic mechanism that has been extensively studied is DNA methylation. 5-methylcytosine (5mC), N6-methyladenine (6mA), and 4-methylcytosine (4mC) are examples of DNA methylation alterations. While 6mA and 4mC are widespread in bacterial genomes, 5mC is the most widely distributed methylation type in eukaryotes, as well as the most researched and understood DNA modification pattern in general. There are varieties of traditional methods for determining the approximate or exact methylation level of DNA.

The majority of DNA methylation experiments use bisulfite modification, which changes cytosine to uracil in single-stranded DNA but has no effect on 5mC. Other methods rely on digesting genomic DNA with specialized endonucleases with varying methylation sensitivities to get a reasonable estimate of total DNA methylation. Over the last few decades, DNA methylation analysis technologies have vastly advanced. Long sequence reading is possible with recently developed third generation sequencing based technologies, which opens up interesting possibilities for studying a wide range of base alterations without bisulfite treatment, such as 5mC, 6mA, and 4mC. Techniques for detecting oxidized versions of 5mC, such as 5-hydroxymethylcytosine (5hmC), have also been developed.

Efforts are currently being made to better understand the epigenetic mechanism and its relationship to a variety of disorders, as well as to uncover epigenetic targets and regulation for possible therapeutic applications. While changes in the gene sequence are often connected with cancer, extensive study has demonstrated the role of epigenetic modifications as well. The predominant epigenetic change is identified, along with histone modification and non-coding RNAs, is altered DNA methylation. Cancer epigenetics has developed into a well-studied area of oncology and genetics. The epigenetic process of DNA methylation, as well as its regulation, is critical for cellular functions to work properly. It affects gene expression, genomic imprinting, and genomic stability, and abnormalities and dysregulation have been seen in a variety of illnesses. In practical all types of cancer cells, abnormal DNA hypermethylation has been identified, and its involvement in carcinogenesis is currently being explored. CpG islands are frequently located within the promoter regions of regulatory genes or exons; they are unmethylated in normal cells, but the 5(C) of the cytosine nucleotide in the CpG islands region is hypermethylated in cancer cells.

DNMTs catalyse the methylation of cytosine at 5(C), with the methyl group coming from the donor S-adenosyl methionine. DNA hypermethylation is commonly associated with transcriptional repression and as a result, loss of tumour suppressor gene function. Hypermethylation was seen in the promoter regions of tumour suppressor genes in general. Hypermethylation of the transcribed region has a mixed effect on gene expression; hypomethylation usually results in the expression of genes that are ordinarily silent. According to study, the polycomb complex, which is responsible for de novo methylases DNMT3A and DNMT3B, which cause hypermethylation in the CpG region, marks the unmethylated CpG islands in various genomes. Ten-Eleven Translocation (TET) and related enzymes, on the other hand, aid in the demethylation of 5(C)-methyl cytosine. Global hypomethylation has been reported in epithelial ovarian cancer. In the instance of breast cancer, many genes have been found to have hypermethylation in the promoter region. The hypermethylation of MLH1 suggests that tumour suppressor gene silencing may hinder the DNA repair mechanism.

CONCLUSION

The epigenetic revolution which has occurred in biology over the last few decades has doubt on the long-held belief that the genetic code is the most important determinant of cellular gene function, and that its change is the primary cause of human diseases. Advances in cancer epigenetics have led to the recognition that the packaging of the genome may be as important as the genome itself in regulating the critical cellular activities required for maintaining cellular identity, as well as in causing diseases like cancer.

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Received: 02-May-2022; Manuscript No. CDB-22-18117; **Editor assigned:** 05-May-2022; Pre QC No. CDB-22-18117 (PQ); **Reviewed:** 19-May-2022; QC No. CDB-22-18117; **Revised:** 26-May-2022; Manuscript No. CDB-22-18117 (R); **Published:** 02-Jun-2022, DOI: 10.35248/2168-9296.22.11.247.

Citation: Diaf K (2022) Determination of DNA Methylation by Bisulfite Modification. Cell Dev Biol. 11:247.