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Cancer Treatment by Targeting Epigenetic Regulators

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Opinion Article

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DESCRIPTION

Epigenetic changes are heritable but reversible changes in histone or DNA modifications that regulate gene function outside of the underlying sequence. Epigenetic dysregulation is frequently linked to human disease, including cancer. Epigenetic-targeted therapy has been used in the treatment of haematological malignancies and has proven realistic therapeutic potential in preclinical and clinical trials for solid tumours since the discovery of several medicines targeting epigenetic regulators.

The study of changes in gene expression that do not result from changes in the DNA sequence is referred to as epigenetics. Chemical alterations in DNA and chromatin can lead to changes in many regulatory processes, resulting in altered gene expression patterns. In some situations, epigenetic markers can be inherited and can alter in response to environmental stimuli throughout the course of an organism's life.

Many diseases have a genetic component, but the epigenetic mechanisms underlying many of them are still being discovered. A large number of disorders are known to alter gene expression within the body, and epigenetic involvement provides a reasonable explanation for how they do so. These alterations may be the source of illness

symptoms. Several diseases, particularly cancer, have been suspected of selectively turning genes on or off, allowing tumorous tissues to evade the host's immune response.

Following the discovery of DNA and the double helix structure, classical genetics has long thought that DNA sequences determine cell behaviours. DNA is bundled into chromatin in cells, with nucleosomes functioning as the basic repeating unit. A 147-base-pair (bp) DNA tract surrounds an octamer of four core histones (H2A, H2B, H3, and H4). Nucleosomes are separated by 10–60 bp DNA. Researchers have gradually identified species that have the same genetic information but exhibit diverse behaviours, such as somatic cells from the same human that share a genome but behave fundamentally differently.

This heritable phenomena includes a wide spectrum of biological processes such as cell proliferation, differentiation, and disease development. DNA methylation, histone modification, histone readout, chromatin remodelling, and the effects of noncoding RNA are all examples of epigenetic events. There are three types of elements involved in various modification patterns: "writer," "reader," and "eraser." Writers and erasers are enzymes that transfer or remove chemical groups from DNA or histones, respectively. Reader proteins can detect changes in DNA or histones.

The epigenome works with other regulatory factors, like as transcription factors and noncoding RNAs, to govern genome expression or repression in order to coordinate various biological functions. External stimuli and cellular signalling networks can potentially influence epigenetics. These impacts are both short-term and long-term. Given the significance of epigenetics in influencing cell functions, a better understanding of both normal and pathological epigenetic processes can help in the study and potential treatment of a variety of disorders, including cancer.

Cancer is caused by a combination of environmental and inherited factors. Genetic information change in cancer cells is frequently apparent. Cancer is characterised by epigenome dysregulation, as well as genome instability and mutation. Some of the modifications have an impact on cell function and contribute to neoplastic transformation. However, the cancer phenotype can be restored to normal by correcting these changes with drugs or gene therapy. Holliday argued that epigenetic changes cause cancer. A variation in the risk of malignant transformation could be explained by a change in cellular methylation status produced by a specific methyltransferase.

Cancer patients with the same stage and grade have very different outcomes in clinical settings. Diverse tumour cells in tumour tissues exhibit diverse patterns of histone modification, either genome-wide or in specific genes, demonstrating the existence of epigenetic heterogeneity at the cellular level. Similarly, the use of molecular biomarkers is viewed as a potential technique for patient classification. It is critical to emphasise that cancer is the product of multiple epigenetic mechanisms working in concert.