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Short Communication

Role of Epigenetic Modifications in Human Cancer

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DESCRIPTION

Epigenetics is the study of heritable and reversible changes in gene expression that occur without altering the underlying DNA sequence. These changes determine how genes are switched on or off and play a crucial role in regulating normal cellular functions, including growth, differentiation, development, and tissue maintenance [1]. Epigenetic regulation ensures that cells with identical genetic material can perform specialized functions by expressing distinct sets of genes. In recent years, epigenetics has become a major focus of biomedical research because abnormal epigenetic modifications are recognized as important contributors to the initiation and progression of numerous diseases, particularly cancer. Unlike genetic mutations, epigenetic alterations are dynamic and potentially reversible, making them valuable targets for disease diagnosis, prevention, and therapy [2].

The regulation of gene expression through epigenetic mechanisms primarily involves Deoxyribonucleic Acid (DNA),histone modifications, and non-coding Ribonucleic Acid (RNA) molecules. DNA methylation occurs through the addition of methyl groups to cytosine bases located within CpG-rich regions of the genome. This process generally suppresses gene transcription by preventing the binding of transcription factors or recruiting proteins that compact chromatin [3]. In healthy cells, DNA methylation contributes to genomic stability, normal embryonic development, and proper regulation of tissue-specific genes. However, abnormal methylation patterns are frequently observed in cancer, where excessive methylation of tumor suppressor gene promoters leads to gene silencing, while global hypomethylation contributes to genomic instability and activation of oncogenes [4].

Histone proteins package DNA into chromatin, and their chemical modifications significantly influence gene accessibility and transcriptional activity. Histone acetylation, methylation, phosphorylation, and ubiquitination regulate whether chromatin exists in a condensed or relaxed state. Histone acetylation generally promotes active gene expression by loosening chromatin structure, whereas deacetylation results in tighter chromatin packing and reduced transcription. Histone methylation may either activate or repress gene expression depending on the specific amino acid residues involved [5]. The coordinated interaction between DNA methylation and histone modifications establishes a complex regulatory network that controls cellular identity and function. Disruption of these regulatory mechanisms has been associated with uncontrolled cell proliferation, impaired apoptosis, and increased susceptibility to malignant transformation [6].

Non-coding RNAs represent another important component of epigenetic regulation. MicroRNAs are small RNA molecules that regulate gene expression by degrading messenger RNA or preventing protein translation. Depending on their target genes, microRNAs may function as oncogenes or tumor suppressors [7,8]. Long non-coding RNAs participate in chromatin remodelling, transcriptional regulation, and RNA processing through interactions with proteins and DNA. Abnormal expression of non-coding RNAs has been linked to cancer progression, metastasis, therapeutic resistance, and altered immune responses. Their stability in blood and other biological fluids has also generated considerable interest in their use as non-invasive biomarkers for early cancer detection and disease monitoring [9,10]. Their expression patterns can provide valuable information about tumor development and patient prognosis. Further research may help establish non-coding RNAs as reliable tools for personalized cancer diagnosis and treatment.

CONCLUSION

In conclusion, epigenetics provides a critical layer of gene regulation that influences cellular behavior without changing the DNA sequence. Its dynamic and reversible nature distinguishes it from genetic mutations and offers unique opportunities for disease prevention, early diagnosis, prognostic evaluation, and therapeutic innovation. Continued advances in epigenetic research will strengthen precision medicine approaches and contribute to more effective strategies for improving human health and reducing the global burden of cancer.

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REFERENCES

1. Thrift AP, Wenker TN, El-Serag HB. Global burden of gastric cancer: epidemiological trends, risk factors, screening and prevention. Nat Rev Clin Oncol. 2023;20(5):338-349.
2. Kondo A, Shinozaki-Ushiku A, Rokutan H, Kunita A, Ikemura M, Yamashita H,et al. Loss of viral genome with altered immune microenvironment during tumour progression of Epstein-Barr virus-associated gastric carcinoma. J Pathol. 2023;260(2):124-136.
3. Fukayama M, Kunita A, Kaneda A. Gastritis-infection-cancer sequence of Epstein-Barr virus-associated gastric cancer. Adv Exp Med Biol. 2018:437-457.
4. Stanland, L.J.; Luftig, M.A. Molecular features and translational outlook for Epstein-Barr virus-associated gastric cancer. Future Virol. 2018;13:803–818.
5. Bitler BG, Aird KM, Garipov A, Li H, Amatangelo M, Kossenkov AV,et al. Synthetic lethality by targeting EZH2 methyltransferase activity in ARID1A-mutated cancers. Nat Med. 2015;21(3):231-238.
6. GTEx Consortium. The GTEx Consortium atlas of genetic regulatory effects across human tissues. Science. 2020 ;369(6509): 1318-1330.
7. Suehnholz SP, Nissan MH, Zhang H, Kundra R, Nandakumar S, Lu C,et al. Quantifying the expanding landscape of clinical actionability for patients with cancer. Cancer Discov. 2024;14(1): 49-65.
8. Horak P, Fröhling S. Measuring progress in precision oncology. Cancer discovery. 2024;14(1):18-19.
9. Qi H, Zhu H, Lou M, Fan Y, Liu H, Shen J,et al. Interferon regulatory factor 1 transactivates expression of human DNA polymerase η in response to carcinogen N-methyl-N′-nitro-N-nitrosoguanidine. J Biol Chem. 2012;287(16):12622-12633.
10. Portela A, Esteller M. Epigenetic modifications and human disease. Nat Biotechnol. 2010;28(10):1057-1068.