Epigenetic Modifications as Predictive Biomarkers in Colorectal Cancer Treatment

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Description

One of the main causes of cancer-related mortality globally is Colorectal Cancer (CRC). The prognosis for advanced colorectal cancer is still not good, despite improvements in screening, early detection and therapy. Personalized medicine has demonstrated potential in enhancing results by customizing care according to unique patient attributes. The use of epigenetic alterations as predictive biomarkers is one area of personalized medicine that is gaining attention. The emergence and spread of cancer are significantly influenced by epigenetic alterations, such as DNA methylation, histone modifications and non-coding RNAs. The potential of epigenetic changes as predictive biomarkers in the treatment of colorectal cancer is examined in this essay, which also discusses their mechanics, clinical uses and future approaches.

Changes in gene expression that is heritable and independent of DNA sequence changes are referred to as epigenetic modifications [1]. Enticing targets for therapeutic intervention, these modifications are reversible and have the ability to control gene activity. Histone changes, DNA methylation and non-coding RNAs are the three primary categories of epigenetic alterations. DNA methylation is the process of adding a methyl group to cytosine residues at their 5' position; this usually occurs in CpG islands close to gene promoters. Tumor suppressor gene promoter hyper methylation can result in gene silence, which advances the development of cancer. On the other hand, hypo methylation can make oncogenes active. Gene transcription is regulated by histone changes that alter chromatin accessibility, such as acetylation, methylation, phosphorylation and ubiquitination. Non-coding RNAs, Including Long Non-Coding RNAs (lncRNAs) and microRNAs (miRNAs), can target mRNA transcripts for translational inhibition or degradation, hence modifying gene expression at the post-transcriptional level. As prognostic biomarkers for colorectal cancer, DNA methylation patterns have been thoroughly investigated. One characteristic of CRC is aberrant DNA methylation; numerous genes with altered methylation status have been found. For example, Microsatellite Instability (MSI), a subtype of Colorectal Cancer (CRC) with a high mutation rate, is linked to hyper methylation of the gene promoter [2]. methylation is a valuable biomarker for predicting treatment response since immune checkpoint drugs are effective in treating MSI-high colorectal cancers. The methylation of the Septin 9 ( ) gene is an additional illustration. Since CRC frequently exhibits hyper methylation of the SEPT9 gene, its identification in blood plasma provides a non-invasive diagnostic for an early diagnosis. Additionally, resistance to specific chemotherapies has been associated with methylation of genes including , APC and , which offers important information for treatment planning [3].

In CRC, histone alterations may also be prognostic indicators. These alterations can change gene expression and chromatin structure, which can impact how quickly cancer progresses and how well a

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and DNA methylation (like DNA methyl transferase inhibitors) are either being studied or used in clinical settings. In order to maximize the effectiveness and reduce the negative effects of these medicines, it may be possible to identify patients who have particular epigenetic modifications [10].

References

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