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Perspective

Epigenetics Marker for Cancer Detection: A Promising Approach

Kim Hariss*

Department of Oncology, University of Manchester, United Kingdom

INTRODUCTION

Cancer is a complex disease that arises from the accumulation of genetic and epigenetic alterations. While genetic mutations can directly alter the DNA sequence, epigenetic modifications can affect gene expression without altering the underlying DNA code [1]. Epigenetic changes, including DNA methylation, histone modification, and non-coding RNA expression, are increasingly recognized as critical drivers of cancer initiation and progression. Epigenetic modifications can be used as biomarkers for early cancer detection, prognosis, and treatment response. In this article, we will discuss the potential of epigenetic markers for cancer detection and their current status in clinical practice [2]. Aberrant DNA methylation, a type of epigenetic modification, is a hallmark of many cancers. Specific DNA methylation patterns can be used as biomarkers for cancer diagnosis, prognosis, and treatment response.

DESCRIPTION

Epigenetic modifications are essential for normal development and cellular differentiation. They regulate gene expression by controlling chromatin structure and accessibility to transcription factors. Aberrant epigenetic changes can lead to altered gene expression and disrupted cellular homeostasis, contributing to cancer development. DNA methylation is the most widely studied epigenetic modification in cancer. It involves the addition of a methyl group to the cytosine residue of CpG dinucleotides, predominantly in the promoter regions of genes [3]. DNA hypermethylation results in gene silencing, while hypomethylation leads to gene activation. Histone modifications, including acetylation, methylation, and phosphorylation, also play a crucial role in gene expression regulation. Histone acetylation is generally associated with active transcription, while histone methylation can either activate or repress gene expression, depending on the specific histone residue and degree of methylation.

Epigenetic changes in cancer can occur early in tumor development, making them potential biomarkers for early detection. DNA methylation is a promising epigenetic marker for cancer detection as it is stable, reproducible, and can be detected in various biological specimens, including blood, urine, and tissue samples. Hypermethylation of tumor suppressor genes, such as CDKN2A (p16), APC, and RASSF1A, has been detected in various cancers, including lung, breast, colon, and prostate cancer. Hypermethylation of these genes can serve as an early indicator of cancer development and can be used for cancer screening and risk assessment. DNA methylation can also be used to distinguish cancer from normal tissues and to identify subtypes of cancer based on their methylation patterns [4].

Histone modifications have also been studied as potential biomarkers for cancer detection. Histone modifications are more dynamic than DNA methylation and can reflect changes in gene expression in real-time. For example, histone acetylation levels are reduced in breast cancer, indicating a loss of gene activation. Conversely, histone methylation is increased in prostate cancer, leading to gene silencing. Non-coding RNA, including microRNA and long non-coding RNA, can also function as epigenetic markers for cancer detection. MicroRNA is a small RNA molecule that can regulate gene expression by binding to target mRNA and promoting degradation or translational repression. Dysregulated microRNA expression has been observed in various cancers, including lung, breast, and pancreatic cancer. Long non-coding RNA can also regulate gene expression by interacting with chromatin and transcription factors. Aberrant expression of long non-coding RNA has been associated with cancer development and progression [5].

CONCLUSION

Despite the promise of epigenetic markers for cancer detection, their clinical application is still limited. One of the major challenges is the lack of standardized detection methods and the variability of results between different laboratories. Current methods for detecting epigenetic markers, including bisulfite sequencing, methylation-specific PCR, and microarray analysis, have limitations in terms of sensitivity, specificity, and cost-effectiveness. Another challenge is the heterogeneity of cancer epigenomes, which can vary depending on the tumor type overall, epigenetic biomarkers hold great promise for improving

Correspondence to: Kim Hariss, Department of Oncology, University of Manchester, United Kingdom; E-mail: khrss610@gmail.com

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diagnostics and personalized medicine. However, more research is needed to fully understand the complex relationships between epigenetic modifications, environmental factors, genetics, and disease.

REFERENCES

1. Stankiewicz P, Sen P, Bhatt SS, Storer M, Xia Z, Bejjani BA, et al. Genomic and genic deletions of the FOX gene cluster on 16q24.1 and inactivating mutations of FOXF1 cause alveolar capillary dysplasia and other malformations. Am J Hum Genet. 2009;84(6):780–791.
2. Slot E, Edel G, Cutz E, van Heijst A, Post M, Schnater M, et al. Alveolar capillary dysplasia with misalignment of the pulmonary veins: clinical, histological, and genetic aspects. Pulm Circ. 2018;8(3).
3. Slot E, von der Thusen JH, van Heijst A, van Marion R, Magielsen F, Dubbink HJ, et al. Fast detection of FOXF1 variants in patients with alveolar capillary dysplasia with misalignment of pulmonary veins using targeted sequencing. Pediatr Res. 2021;89(3):518–525.
4. Szafranski P, Gambin T, Dharmadhikari AV, Akdemir KC, Jhangiani SN, Schuette J, et al. Pathogenetics of alveolar capillary dysplasia with misalignment of pulmonary veins. Hum Genet. 2016;135(5):569–586.
5. Szafranski P, Dharmadhikari AV, Wambach JA, Towe CT, White FV, Grady RM, et al. Two deletions overlapping a distant FOXF1 enhancer unravel the role of lncRNA LINC01081 in etiology of alveolar capillary dysplasia with misalignment of pulmonary veins. Am J Med Genet A. 2014;164A(8):2013–2019.