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Epigenetic Modulation of Important Receptor Pathways

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Introduction

Epigenome and genes help cancer grow and progress. The study of heritable changes in gene expression without a change in DNA sequence is called epigenetics. Reversible epigenetic changes include DNA methylation, chromatin modifications, nucleosome placement, and changes in noncoding RNA profiles. When epigenetic mechanisms are disrupted, gene function can be altered and cellular neoplastic transformation can take place. Before genetic changes, epigenetic alterations typically occur early in the neoplastic process. Epigenetic biomarkers for disease detection, prognosis, risk assessment, and monitoring have been discovered and their underlying epigenetic modifications during carcinogenesis have been better understood thanks to recent technological advancements. We focus on epigenetic alterations that cause genetic changes and the future clinical implications of epigenetic research in this chapter, which focuses on several epigenetic processes and their role in carcinogenesis [1].

Description

Recent research has shown that epigenetics plays a crucial role in the development of cancer. It has been demonstrated that numerous cancer models have abnormal DNA and histone modifications that either activate oncogenes or mutate genes that suppress tumors. While epigenetics has been shown to play a role in some solid tumor cancers, like colon cancer, there is growing evidence that it also plays a role in breast and prostate cancer. Patterns of DNA methylation have been linked to the status of hormone receptors and the growth of breast cancer tumors. In prostate cancer, epigenetic changes have also been linked to androgen receptor status and treatment responsiveness. Unraveling the mechanisms and potential targets of epigenetic modulation of important receptor pathways and activities, which alter clinical therapeutic treatment choices, is this research's top priority. In order to evaluate epigenetic modifications and provide the cutting-edge tools necessary for such research, a new set of methylation arrays has been released. Nutritional therapies that alter epigenetic changes hold particular promise [2].

Researchers will be able to develop translational applications that can be utilized as biomarkers for risk and prognosis, as well as therapeutic possibilities, if they have a solid understanding of the causes and effects of epigenetic modifications. When people change their lifestyles and how they eat, they are more likely to get diseases and cancers related to their diet. Changes in diet have also been shown to significantly lower illness risk. Nutrigenomics is a relatively new field with a lot of potential for treating and preventing certain diseases and cancers. Scientists and medical professionals will gain a better understanding of the role that nutrigenomics plays in preventing food and diseases caused by bad habits, like cancer [3].

It has an effect on people's health and susceptibility to illness by determining the metabolic response and gene expression. Epigenetic changes can have an impact on disease incidence and pathogenesis. DNA methylation and chromatin remodelling are the epigenetic processes that occur the most frequently. Omega-3 fatty acids are the best example of nutrition and gene interaction that does not involve DNA methylation, despite the fact that certain bioactive food chemicals have been shown to have an effect on cancer prevention via an epigenetic mechanism. Dietary polyphenols can partially prevent oral, breast, cutaneous, oesophageal, colorectal, prostate, pancreatic, and lung cancers [4].

Vitamins and minerals are also involved in processes that regulate. Anticancer properties can be found in zinc, selenium, and folate, all of which are involved in DNA repair. Taking a multivitamin prevents cancer cells from becoming methylated. A specific population of cells known as "cancer stem cells," which have been regarded as a powerful driving force of carcinogenesis and a fundamental mechanism of treatment resistance, can emerge from mass tumors, according to evidence. Recent advances in epigenomics have revealed important pathways that are involved in the development of cancer. Epigenetic regulation plays a role in cancer development. In this review, we investigate how dysregulation of numerous epigenetic mechanisms may contribute to the onset and progression of cancer, particularly with regard to the maintenance and survival of cancer stem cells [5].

Conclusion

New avenues for identifying cancer stem cells and improving cancer treatment in general are provided by this information in addition to the outcomes of numerous promising clinical and preclinical studies of epigenetic modifying medications. The most prevalent form of cancer in the world is colorectal cancer (CRC). Adenocarcinomas are the result of a buildup of genetic and epigenetic changes in colon epithelial cells. Cancer epigenetics has made significant progress in the past ten years, particularly with regard to abnormal DNA methylation. The epigenome of colon cancer has been studied, and it has been found that nearly all CRCs have genes that are incorrectly methylated. The typical CRC methylome contains hundreds to thousands of genes that are incorrectly methylated.

Acknowledgement

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Conflict of Interest

None.

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