Brief Report
Volume 6:2, 2022

Journal of Genetics and DNA Research

Open Access

All about Epigenetics in Cancer and its Prevention

Yanyuan Wu*

Department of Surgery, University of Washington, USA

Introduction

Cancer development and progression are aided by epigenetic and genetic changes. Epigenetics is the study of heritable changes in gene expression that occur without a change in DNA sequence. DNA methylation, chromatin modifications, nucleosome placement, and changes in noncoding RNA profiles are all examples of reversible epigenetic changes. Gene function can be changed and cellular neoplastic transformation can occur when epigenetic mechanisms are disrupted. Epigenetic alterations occur before genetic changes, and they generally happen early in the neoplastic process. Recent technical advancements have provided insight into the development of possible epigenetic biomarkers for detection, prognosis, risk assessment, and disease monitoring, as well as a better understanding of the underlying epigenetic modifications during carcinogenesis.

In this chapter, we discuss several epigenetic processes and their function in carcinogenesis, with a focus on epigenetic alterations that cause genetic changes, as well as the future clinical implications of epigenetic research. Epigenetics has been discovered as a crucial player in cancer genesis in recent studies. Multiple cancer models have been shown to contain abnormal DNA and histone alterations that mute tumour suppressor genes or activate oncogenes. While epigenetics has a well-established involvement in certain solid tumour malignancies, such as colon cancer, there is growing evidence that epigenetics is also important in breast and prostate cancer. DNA methylation patterns have been related to hormone receptor status and tumour growth in breast cancer. Epigenetic alterations have also been linked to androgen receptor status and treatment responsiveness in prostate cancer.

Description

Epigenetic modulation of important receptor pathways and activities, which alter clinical therapeutic treatment choices, makes unravelling mechanisms and prospective targets a top priority in this research. A new set of methylation arrays has been released to assess epigenetic modifications and give the cutting-edge tools needed to do such research. Nutritional therapies that impact epigenetic modifications offer special promise. In the end, understanding the causes and consequences of epigenetic modifications will help researchers develop translational applications that may be used as biomarkers for risk and prognosis, as well as therapeutic possibilities.

People are more at risk of diet-related illnesses and malignancies when their lifestyles and eating habits change. Dietary changes have also been shown to greatly lessen the risk of illness. Nutrigenomics is a relatively new field, but it has a lot of promise for the prevention and management of certain cancers and disorders. This review will help scientists and health professionals better understand the function of Nutrigenomics in the prevention of food and lifestyle-related disorders like cancer. By determining the metabolic response and gene expression, it has an impact on people's health and illness susceptibility.

Disease incidence and pathogenesis can be influenced by epigenetic changes. The most frequent epigenetic processes are DNA methylation and chromatin remodelling. While certain bioactive food chemicals have a documented impact in cancer prevention through an epigenetic mechanism, omega 3 fatty acids are the finest example of nutrition and gene interaction that does not involve DNA methylation. Oral, breast, cutaneous, oesophageal, colorectal, prostate, pancreatic, and lung cancers are all prevented in part by dietary polyphenols. Minerals and vitamins are also involved in regulatory processes. Zinc, selenium, and folate, all of which are involved in DNA repair, have anticancer effects.

Multivitamin supplementation stops cancer cells from becoming methylated. Evidence has shown that mass tumours can emerge from a specific population of cells known as "cancer stem cells," which have been considered as a powerful driving force of carcinogenesis and a fundamental mechanism of treatment resistance. Epigenetic regulation contributes to cancer development, and recent breakthroughs in epigenomics have shed light on crucial pathways [1-5].

We examine how dysregulation of numerous epigenetic mechanisms might contribute to cancer start and carcinogenesis, particularly in terms of cancer stem cell maintenance and survival, in this review. This information, together with the results of numerous promising clinical and preclinical studies of epigenetic modifying medications, opens up new avenues for identifying cancer stem cells and enhancing cancer treatment in general. Colorectal cancer (CRC) is the most common kind of cancer in the world. It is caused by a build-up of genetic and epigenetic alterations in colon epithelial cells, resulting in adenocarcinomas.

Over the last decade, significant progress has been achieved in the field of cancer epigenetics, notably in the area of abnormal DNA methylation. The epigenome of colon cancer has been studied, and it has been discovered that nearly all CRCs have inappropriately methylated genes, with the typical CRC methylome having hundreds to thousands of incorrectly methylated genes [1-5].

Conclusion

A subset of these methylation genes, known as driver genes, is thought to play a functional role in CRC, similar to gene mutations in the cancer genome. The analysis of methylated genes in CRCs also showed a distinct molecular subtype of CRCs known as CpG island methylator phenotype (CIMP) malignancies, which had a high frequency of methylated genes. Epigenetic changes are being explored as clinical biomarkers for diagnostic, prognostic, and therapeutic uses as a result of breakthroughs in our knowledge of aberrant methylation in CRC. Progress in this research predicts that epigenetic modifications will be widely employed to direct CRC prevention and therapy in the near future.

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***Address for Correspondence:** Yanyuan Wu, Department of Surgery, University of Washington, USA, E-mail: Wuy@yahoomail.com*

Received: 01 March, 2022, Manuscript No. jgdr-22-59088; **Editor assigned:** 03 March, 2022, PreQC No. P-59088; **Reviewed:** 07 March, 2022, QC No. Q-59088; Revised: 12 March, 2022, Manuscript No. R-59088; **Published:** 18 March, 2022, DOI: 10.37421/jgdr.2022.6.119

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How to cite this article: Wu, Yanyuan. "All about Epigenetics in Cancer and its Prevention." J Genet DNA Res 6 (2022): 119.