Abstract:

Epigenetics refers to stable alterations in gene expression with no underlying modifications in the genetic sequence and is best exemplified by differentiation, in which multiple cell types diverge physiologically despite a common genetic code. Interest in this area of science has grown over the past decades, especially since it was found to play a major role in physiologic phenomena such as embryogenesis, imprinting, and X chromosome inactivation, and in disease states such as cancer. The latter had been previously thought of as a disease with an exclusive genetic aetiology. However, recent data have demonstrated that the complexity of human carcinogenesis cannot be accounted for by genetic alterations alone, but also involves epigenetic changes in processes such as DNA methylation, histone modifications, and microRNA expression. In turn, these molecular alterations lead to permanent changes in the expression of genes that regulate the neoplastic phenotype, such as cellular growth and invasiveness. Targeting epigenetic modifiers has been referred to as epigenetic therapy. The success of this approach in hematopoietic malignancies validates the importance of epigenetic alterations in cancer, not only at the therapeutic level but also with regard to prevention, diagnosis, risk stratification, and prognosis.

Human cancers are initiated and maintained by DNA mutations and also by alterations in the cancer epigenome. The interaction between these processes is underscored by the observation that all human cancers contain DNA methylation aberrations and also frequently harbor mutations in genes that modify the epigenome. These include the enzymes that apply and remove methyl groups to and from cytosine, *DNMT3A* and *TET2*. Abnormal DNA cytosine methylation has received the most attention in our understanding of its potential to silence tumour suppressor genes and thus to directly contribute to the malignant phenotype. There are now many drugs that can target the epigenome, and I will focus on those that inhibit DNA methylation. It is important that we understand how these drugs elicit responses in patients, and most of the attention to date has centred on their ability to activate genes that are methylated and altered in a cancer relative to the normal tissue. However, a large percentage of the human genome codes for transposable elements, and these are often silenced by DNA methylation. Activation of these transposable elements can give rise to a state of “viral mimicry” in which a cancer cell behaves as though it is infected by an exogenous virus. I will explore how this activation of transposable elements may contribute to an interferon response that leads to apoptosis of cancer cells and to increased visibility to the immune system. Coupling epigenetic therapies with other approaches may be useful in the future in the treatment of cancers, including bladder cancer.

Abstract Made by: A KATHIR KARUPPIAH

Contact No: +91 9444338218 (WhatsApp Available)

E-Mail Id: kathirkaruppiah@yahoo.com

Currently Pursuing MBBS From Panimalar Medical College Hospital & Research Institute, Chennai. Tamil Nadu, India

From: Chennai, Tamil Nadu, India

Abstract made to: 5TH International Conference on Epigenetics & Cancer, Barcelona, Spain