Cancer epigenetics in Human Disease

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Abstract (300 word limit)

DNA methylation, histone modification, and microRNA (miRNA) are examples of epigenetic mechanisms that can cause heritable phenotypic changes without changing the DNA sequence. A disruption of gene expression patterns mediated by epigenetics can lead to autoimmune illnesses, cancer, and a variety of other ailments. DNA methylation (and demethylation), histone changes, and non-coding RNAs like microRNAs are all epigenetic mechanisms. In comparison to the numerous studies that have concentrated on the topic of genetics, epigenetics research is relatively new. Unlike genetic alterations, which are difficult to erase, epigenetic aberrations can be reversed with pharmaceuticals. Epigenetic indicators can be employed as preventive, diagnostic, and therapeutic markers, according to new research. Drugs that target the precise epigenetic pathways involved in gene expression regulation are currently being developed. In the foetus, the heart is the first organ to function. The creation of the heart is a complex morphogenetic process that is influenced by both genetic and epigenetic factors. The most common congenital disorders are congenital heart diseases (CHD). Genetics alone is insufficient to explain these disorders and their consequences for patients. Epigenetics is becoming more widely recognised as a cause of heart abnormalities. This review provides the most up-to-date information on heart development biology. It also gives a general overview of epigenetics, with a focus on chromatin's three-dimensional conformation. Then, we review what we now know about the role of epigenetics in cardiac cell fate determination. We also present an update on the role of epigenetic abnormalities in the development of CHD. Epigenetic processes regulate gene activity and organism development. DNA methylation, histone modifications, and RNA-mediated activities are all part of the epigenome, and disrupting this equilibrium can lead to a variety of diseases, including obesity and type 2diabetes (T2D). In connection to obesity and T2D, this Review highlights epigenetic fingerprints found from human tissues relevant to metabolism, such as adipose tissue, skeletal muscle, pancreatic islets, liver, and blood. Despite the fact that this field of study is still in its infancy, these comprehensive findings support not just an epigenetic role in illness development, but also epigenetic modifications as a response to disease. Epigenetic variability is influenced by genetic predisposition as well as ageing, and various environmental factors, such as exercise and diet, interact with the human epigenome. Cancer initiation 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 changes occur before genetic changes, and they usually happen early in the neoplastic process. Recent technical advancements have provided insight into the discovery 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. DNA methylation is one of the most common and well-studied epigenetic alterations, and it is critical for normal development and cellular life. Changes in the global DNA methylation landscape lead to changes in the transcriptome and cellular pathway dysregulation. Improved technologies for studying DNA methylation patterns and dynamics at base pair resolution and across individual DNA molecules on a genome-wide scale have revealed the breadth of change in the DNA methylation landscape in disease states, especially during carcinogenesis. More

recently, DNA hydroxymethylation profiling techniques have been developed, which allow for the discrimination of 5mC and 5hmC profiles and provide additional insights into DNA methylation dynamics and remodelling in cancer. In this study, we look at how DNA methylation and DNA hydroxymethylation are distributed in distinct genomic contexts, first in normal cells and then in cancer. Finally, we go over DNA methylation profiling techniques, including single-cell methods, bisulfite-free methods, and ultra-long read sequencing techniques.

Important of research

The intensive research being carried out in the field the study of epigenetic modifications in normal and diseased tissues has progressed significantly. Epigenetic research has been concentrated on cancer thus far, but as the discipline has progressed, new insights into other diseases, particularly neurological and autoimmune diseases, have emerged. Epigenetic changes are likely to be found in other diseases; indeed, they have already been identified in cardiovascular diseases, metabolic diseases, myopathies, and children born through assisted reproductive technologies. The description of full DNA methylomes of humans and viruses, the possible discovery of non-CpG methylation, the defining of CpG island shores, and so on has all been made in recent months. the role of abnormal DNA methylation in illnesses other than cancer, the definition of new histone modifications and variants, as well as their functions, the discovery of new epigenetic machinery such as the DNA demethylase Tet1 and the histone kinase JAK2, the description of new epigenetic mutations, and the flurry of ncRNA studies emphasising the relevance of RNA-mediated epigenetic regulation. Epigenomic analysis on a broad scale is now possible thanks to technological advancements. The first whole-genome, high-resolution epigenetic maps are now available, but we shouldn't stop there. In both healthy and pathological tissues, detailed human DNA methylomes, histone modification, and nucleosome positioning maps are required. Several worldwide projects and initiatives have been launched in this regard: among them, the NIH Roadmap Epigenomics Program, the ENCODE Project, the A HEAD Project, and the Epigenomics NCBI browser (see Bernstein and colleagues and Satterlee and colleagues in this issue's commentary).

Biography

Michel Andelfinger has her expertise in evaluation and passion in improving the health and wellbeing. Her open and contextual evaluation model based on responsive constructivists creates new pathways for improving healthcare. She has built this model after years of experience in research, evaluation, teaching and administration both in hospital and education institutions. The foundation is based on Third generation evaluation (Khosla & Jones, 1989) which is a methodology that utilizes the previous generations of evaluation: measurement, description and judgment. It allows for value-pluralism. Professor working in the field of Human disease Completed studies from University School of Medicine, USA. Research interests include medical science, Public health, Welfare of society. This approach is responsive to all stakeholders and has a different way of focusing. Dr. Michel Andelfinger was invited to be the advisory board and keynote speaker for advanced level training of various international companies and courses to train the advanced uses of filler, botox, and thread lift, and invited to be the speaker for Human disease and cancer epigenetics. Michel MBChB, MD, Ph.D. has both medical and biological backgrounds. After 12 years practice of internal medicine in Southeast University, full time and Circulating histones are mediators of cancer injury.

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Importance's of institute

Stanford University School of Medicine is Stanford University's medical school, located in Stanford, California. Its origins can be traced back to the University of the Pacific's Medical Department, which was founded in San Francisco in 1858. In 1908, Stanford purchased this medical school, which was then known as Cooper Medical College. In 1959, the medical school relocated to the Stanford campus in Palo Alto, California. Stanford Medicine includes the School of Medicine, Stanford Health Care, and Lucile Packard Children's Hospital. Stanford Health Care was named California's fourth finest hospital (behind UCLA Medical Center, Cedars-Sinai Medical Center, and UCSF Medical Center, respectively). In the aftermath of the California Gold Rush, Illinois physician Elias Samuel Cooper relocated to San Francisco in 1855. Cooper created the Medical Department of the University of the Pacific, the first medical school on the West Coast, in 1858 on Mission Street near 3rd Street in San Francisco, in collaboration with the University of the Pacific (also known as California Wesleyan College). Cooper died in 1862, and the Medical Department of the University of the Pacific went into decline without him. Cooper's nephew, Levi Cooper Lane, reactivated and reorganized the medical department at the University of the Pacific in 1870, and Lane provided a new building at the corner of Webster and Sacramento Streets in 1882, establishing the department as a separate school, the Cooper Medical College. Lane developed a hospital and a nursing school, as well as making plans for the Lane Medical Library.

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