
Preface

Cancer is a genetic and epigenetic disease. Both genetic and epigenetic changes occur simultaneously in an organism. Genetic alterations include mutations and single-nucleotide changes, deletions, insertions, changes in copy number, and translocations. Epigenetic alterations (methylation, histone and selected nonhistone protein alterations, noncoding RNA alterations, imprinting, and chromatin remodeling) broadly include non-genetic alterations that are capable of being transmitted from one cell generation to another. The epigenetic regulation plays an important role in normal development and maintenance of tissue-specific genes' expression in humans and the disturbance of these patterns lead to changes involved in tumor formation. Global epigenetic changes and genes' promoter-specific methylation patterns have been observed in many cancer types and play an essential role in carcinogenesis. Epigenetic changes have been observed in early stages of tumor development and together with the genetic alterations have been defined as abnormalities, necessary for cancer initiation and progression. Changes in histone modification patterns and microRNA expression also evolved as being important players in the carcinogenic process. Different cancer types express distinct methylation patterns but also share common epigenetic changes that are very important in early detection, progression, and prognosis as well as the design of new therapeutic tools against cancer cells. The technology available to detect these epigenetic changes is evolving rapidly and provides more understanding of these processes in normal and cancerous cells. Several of these technologies are discussed in this book. Recent studies identified several factors that may play a significant role in the initiation of the epigenetic changes in cancer. Some of the genetic and environmental factors that have been shown to be involved in these processes are also discussed.

The epigenome is dynamic and very susceptible to environmental changes. Toxicoepigonomics studies are conducted to explain the epigenetic effects of environmental exposures. Epigenetic programming occurs during development and reflects altered gene expression in disease states. This programming differs from genetic polymorphisms or mutations because genetic changes are reflected in all cells, whereas epigenetic changes are cell and tissue specific. Studies that involve the measurement of epigenetic changes that occur at the genome-wide level and their association with disease are called epigenome-wide association studies (EWAS). Epigenetic alterations respond quickly to environmental changes, and technologies are available to measure these changes. During the last 5 years, excellent progress has been made in the field of altered epigenomic profiling in response to toxins and environmental pollutants. Epigenetic marks are tissue specific; genomic marks are not. This fact has implications in tissue-specific toxicity, pharmacokinetics, and pharmacotoxicity. Genetic marks are static and can be measured at any point; epigenetic marks must be measured at different time points. Epigenetic marks contribute to the phenotypic characteristics of cells, tissues, and persons. The latest in cancer toxicoepigonomics is covered in the book.

All these epigenetic alterations can be used in clinical practice as biomarkers of early cancerous lesions or markers of progression and prognosis. Furthermore, epigenetic

inhibitors have potential in cancer treatment. FDA-approved epigenetic inhibitors and nutrients with chemoprevention potential are discussed.

The book is divided into four parts.

Part I. Background: Epigenetic mechanism.

Part II. Cancer specific type epigenetic changes.

Part III. Methods and technologies used for detecting epigenetic changes.

Part IV. Factors that influence epigenetic changes in cancer.

Different chapters covered in these parts provide the most up-to-date knowledge of epigenetics and its implication in cancer prevention by risk assessment and screening and cancer control by treatment.

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