# Altered primary chromatin structures and their implications in cancer development

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Cancer development is a complex process involving both genetic and epigenetic changes. Genetic changes in oncogenes and tumor-suppressor genes are generally considered as primary causes, since these genes may directly regulate cellular growth. In addition, it has been found that changes in epigenetic factors, through mutation or altered gene expression, may contribute to cancer development.

In the nuclei of eukaryotic cells, DNA and histone proteins form a structure called chromatin that consists of nucleosomes that, like beads on a string, are aligned along the DNA strand. Modifications in chromatin structure are essential for cell type-specific activation or repression of gene transcription, as well as other processes such as DNA repair, DNA replication and chromosome segregation.

In this review, Angelo Ferraro of Kazan Federal University focuses on recently published work dealing with alterations in the primary structure of chromatin resulting from imprecise arrangements of nucleosomes along DNA, and its functional implications for cancer development.

While single aspects of chromatin architecture are reported daily, no comprehensive review has yet been published that summarizes mechanisms such as chromatin remodelling, histone modification, histone variant and nucleosome positioning in cancer.

Alterations in epigenetic factors involved in chromatin dynamics may accelerate cell cycle progression and, ultimately result in malignant transformation. Abnormal expression of remodeler and modifier enzymes, as well as histone variants, may confer to cancer cells the ability to reprogram their genomes and to yield, maintain or exacerbate malignant hallmarks. At the end, genetic and epigenetic alterations that are encountered in cancer cells may culminate in chromatin changes that may, by altering the quantity and quality of gene expression, promote cancer development.

The primary chromatin structure is regulated by a variety of epigenetic mechanisms that may be deregulated through gene mutations and/or gene expression alterations. In recent years, it has become evident that changes in chromatin structure may coincide with the occurrence of cancer hallmarks.

The functional interrelationships between such epigenetic alterations and cancer development are just becoming clear; therefore, the oncology community should continue to explore the molecular mechanisms governing the primary chromatin structure, both in normal cells and cancer cells, in order to improve future approaches for cancer detection, prevention and therapy, as also for circumventing drug resistance.

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