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Eukaryotic Cells Induced by Transcriptional Inhibition Localize Active Chromatin in the Nucleolus

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DESCRIPTION

In eukaryotic cells, proteins and RNA factors involved in genomic activities such as transcription, RNA processing, DNA replication, and repair accumulate in chromatin sub compartments that lack self-assembling membranes. These structures help efficiently carry out chromatin-mediated reactions and establish specific cellular programs. However, the mechanisms underlying their formation are only partially understood. Recent studies have utilized Liquid-Liquid Phase Separation (LLPS) of proteins and RNA in establishing chromatin activity patterns. At the same time, chromatin folding within the cell nucleus may facilitate the partitioning of the genome into spatially distinct domains. Here, we describe the interplay between chromatin organization, chromatin binding and LLPS by comparing and contrasting three typical chromatin sub compartments. Nucleolus, cluster of active RNA polymerase, and centromere heterochromatin domains. We discuss how different pathways of chromatin compartmentalization are related to transcriptional regulation, targeting of soluble factors to specific parts of the genome and disease-causing genetic aberrations. Cells and tissues generate and are subject to different mechanical forces acting at different scales, from tissue to cells to organelles. Force provides important signals that inform developmental cell behavior and adult tissue homeostasis, and alterations in force and its downstream mechanotransduction pathways can influence disease progression. Recently, progress has been made in understanding the mechanisms by which forces regulate chromatin organization and state, and the mechanosensitive transcription factors that coordinate gene expression, cellular state, and behavior in response to the physical properties of the cellular microenvironment. These findings highlight the importance of mechanosensitive transcriptional regulation for physiology, disease, and novel therapeutics. The past decade has witnessed the emergence of sequence-based methods for understanding chromosome organization. The confluence of *in situ* approaches that gather information about loops, topological domains, and larger chromatin compartments enables our understanding of chromatin-associated diseases. Interestingly, recent advances in single-molecule imaging with the ability to reconstruct chromosomal conformational 'mass cell' features have revealed cell-by-cell structural changes in chromatin. The basic questions that motivate the analysis of the literature are can altered chromatin structure drive tumorigenesis? As our community learns more about rare diseases such as cancer with low mutation frequencies, understanding 'chromatin-driven' pathologies will help us understand the genome. The regulatory structure describe recent insights into genomic structural alterations in human cancers and highlight multiple pathways to perturbations of chromatin structure, including structural alterations, non-coding mutations, metabolism, and *de novo* mutations to structural regulators themselves. Genomic architecture characteristic of different classes of chromatin-driven tumors. As we begin to integrate insights from single-cell imaging studies and chromatin structural sequencing, we can begin to understand cellular diversity within common diagnostics and begin to define structure-function relationships in misfolded genomes. In eukaryotes, three-dimensional (3D) chromatin architecture maintains genome stability and is critical for regulating gene transcription. However, little is known about the mechanisms by which various ATP-dependent chromatin remodeling complexes regulate her 3D chromatin structure in plants.

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CONFLICTS OF INTERESTS

The authors declare that they have no conflict of interest.

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