OPEN ACCESS Freely available online

Fungal Genomics & Biology

ISSN: 2165-8056

Perspective

Chromatin-Driven Genome Plasticity in Filamentous Fungi: An Epigenomic Perspective

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DESCRIPTION

Genome plasticity enables filamentous fungi to rapidly adapt to environmental fluctuations, host interactions, and ecological competition. While genetic mutations and recombination events have traditionally been considered the primary boosters of fungal evolution, epigenetic regulation is increasingly recognized as a central determinant of genome function and adaptability. Chromatin remodeling, histone modifications, and higher-order genome organization collectively regulate gene expression, transposable element activity, and secondary metabolism. This article explains how chromatin-based mechanisms shape genome plasticity in filamentous fungi and discusses their implications for pathogenicity and metabolic innovation.

Filamentous fungi occupy diverse ecological niches ranging from soil ecosystems to plant and animal hosts. Their survival depends on rapid transcriptional reprogramming in response to nutrient limitation, temperature shifts, oxidative stress, and host-derived signals. Unlike permanent genetic mutations, chromatin-mediated regulation provides a reversible mechanism for altering gene expression patterns without modifying DNA sequences. Genome plasticity refers to the capacity of an organism to modify genome function and structure in response to environmental pressures. In fungi, this plasticity is strongly influenced by epigenetic modifications that control chromatin accessibility and transcriptional activity.

Histone proteins package Deoxyribonucleic Acid (DNA) into nucleosomes, forming chromatin. Post-translational modifications of histone tails such as methylation and acetylation determine whether chromatin adopts an open (euchromatic) or condensed (heterochromatic) configuration. In many filamentous fungi, methylation of histone H3 lysine residues is associated with transcriptional repression, particularly in repeat-rich genomic regions. This regulation contributes to genome compartmentalization. Many fungal genomes exhibit a bipartite structure composed of gene-dense core regions and repeat-rich accessory regions. Core regions typically contain essential housekeeping genes and display active chromatin marks. In contrast, accessory compartments often harbor transposable elements, effector genes, and secondary metabolite clusters that are frequently epigenetically silenced. Such compartmentalization allows fungi to maintain genome stability while retaining evolutionary flexibility in specific genomic zones. Under stress conditions, chromatin remodeling may relax repression in accessory regions, enabling rapid activation of adaptive genes.

Adenosine Triphosphate (ATP)-dependent chromatin remodeling complexes reposition nucleosomes to regulate promoter accessibility. These complexes respond dynamically to environmental cues, enabling coordinated activation of gene networks involved in development, stress response, and pathogenicity. For example, during host infection, filamentous pathogens often undergo transcriptional reprogramming that activates virulence-associated genes. Chromatin remodeling facilitates this transition by altering nucleosome positioning around effector gene promoters. Because these changes are reversible, fungi can fine-tune gene expression according to host immune pressures. Chromatin remodeling also plays a role in developmental transitions such as sporulation and hyphal differentiation. These processes require precise temporal gene regulation orchestrated by epigenetic mechanisms.

Transposable Elements (TEs) constitute a substantial portion of many fungal genomes. While TE mobilization can generate genetic diversity, uncontrolled activity threatens genome integrity. Epigenetic silencing mechanisms, including histone methylation and Ribonucleic Acid (RNA)-mediated pathways, maintain TE repression under normal conditions. However, environmental stress may partially relieve this repression, allowing limited transposition. Such stress-induced mobilization can create beneficial genetic variation that enhances adaptation. TE insertions may also alter local chromatin states, influencing nearby gene expression. This dual role genome protection and controlled diversification illustrates how chromatin plasticity balances stability and innovation.

Beyond linear sequence organization, fungal genomes are structured within the nucleus in defined spatial configurations. Chromosomal domains interact through long-range contacts that influence transcriptional regulation. Secondary metabolite

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Received: 28-Nov-2025, Manuscript No. FGB-25-40965; **Editor assigned:** 01-Dec-2025, PreQC No. FGB-25-40965 (PQ); **Reviewed:** 15-Dec-2025, QC No. FGB-25-40965; **Revised:** 22-Dec-2025, Manuscript No. FGB-25-40965 (R); **Published:** 29-Dec-2025, DOI: 10.35248/2165-8056.25.15.307

Citation: Sen A (2025). Chromatin-Driven Genome Plasticity in Filamentous Fungi: An Epigenomic Perspective. Fung Genom Biol. 15:307.

gene clusters, for instance, may occupy specific nuclear territories that promote coordinated expression. Changes in environmental conditions can alter three-dimensional genome architecture, modifying interactions between regulatory elements and target genes. Such structural reorganization adds an additional regulatory layer to fungal genome plasticity. Chromatin-mediated regulation directly impacts fungal virulence. Many pathogenic fungi rely on epigenetically controlled effector genes to suppress host defenses. Disruption of chromatin modifiers often attenuates virulence, highlighting potential antifungal targets. From a biotechnological perspective, epigenetic manipulation can activate cryptic secondary metabolite clusters, leading to discovery of novel natural products. Chemical inhibitors of histone deacetylases, for example, have been used to induce production of previously silent compounds.

CONCLUSION

Chromatin plasticity is a fundamental regulator of fungal genome adaptability. Through dynamic histone modifications, nucleosome repositioning, TE regulation, and spatial genome organization, filamentous fungi achieve rapid and reversible transcriptional reprogramming. These mechanisms allow fungi to balance genome stability with evolutionary flexibility, ensuring survival in fluctuating environments. Continued integration of epigenomics, functional genomics, and systems biology will further elucidate how chromatin dynamics shape fungal evolution and pathogenicity.