

Telomere Maintenance and its Role in Fungal Genome Stability

Elena Markovic*

Department of Fungal Biology and Systems Genomics, University of Belgrade, Belgrade, Serbia

DESCRIPTION

Telomeres, repetitive Deoxyribonucleic Acid (DNA) sequences capping chromosome ends, are critical for maintaining genome integrity and regulating cellular lifespan. In fungi, telomere biology influences genome stability, chromosomal rearrangements, and adaptation to environmental stress. Telomerase, recombination-based alternative lengthening mechanisms, and associated telomere-binding proteins coordinate telomere maintenance. This article examines fungal telomere structure, maintenance strategies, their roles in genome evolution, and implications for stress tolerance and pathogenicity.

Chromosomal ends are inherently unstable, prone to degradation, fusion, and incomplete replication. Telomeres solve this problem by providing repetitive DNA sequences bound by protective proteins. In fungi, as in other eukaryotes, telomeres prevent chromosomal end-to-end fusions, maintain replication fidelity, and serve as platforms for genome regulation. Beyond structural protection, telomeres act as regulators of replicative lifespan and genome plasticity. Variation in telomere length and maintenance mechanisms can influence adaptation to environmental stressors and contribute to fungal evolution. Fungal telomeres typically consist of short tandem repeats (e.g., TTAGGG or variants thereof) bound by telomere-specific proteins forming a protective cap. These sequences vary in length among species and even among strains, reflecting evolutionary flexibility. Telomere-binding proteins stabilize chromosome ends and mediate interactions with telomerase. They also participate in subtelomeric chromatin organization, which affects the expression of nearby genes, including secondary metabolite clusters and virulence factors.

Telomerase is a ribonucleoprotein complex that adds repeat sequences to chromosome ends, compensating for the end-replication problem. Fungal telomerase activity varies across species and developmental stages. Mutations in telomerase components often lead to progressive telomere shortening, genomic instability, and reduced replicative capacity. Some fungi employ recombination-based mechanisms to maintain telomere length in the absence or reduction of telomerase activity.

Alanine Aminotransferase (ALT) involves homologous recombination between telomeric sequences, leading to length heterogeneity. This flexibility can contribute to adaptation under stress or during host colonization. Telomere dysfunction triggers DNA damage responses and can result in chromosomal fusions, translocations, or aneuploidy. Controlled instability, however, may provide evolutionary advantages by generating genetic diversity without affecting essential core genes. Subtelomeric regions often harbor rapidly evolving gene families, including virulence effectors, transporters, and secondary metabolite clusters. Telomere maintenance indirectly regulates these adaptive regions, linking structural chromosome integrity with ecological flexibility.

Environmental stresses, such as oxidative damage, nutrient limitation, or thermal fluctuations, accelerate telomere shortening. Fungi respond through upregulation of telomerase or activation of ALT pathways to preserve chromosome stability. Telomere length variation under stress conditions may also influence gene expression in subtelomeric regions, allowing dynamic adjustment of adaptive traits. This mechanism represents a form of reversible genome plasticity mediated by telomere biology. In pathogenic fungi, telomere integrity is critical for maintaining virulence. Efficient telomere maintenance ensures proper chromosome segregation during rapid growth in host tissues. Additionally, subtelomeric positioning of effector genes allows their epigenetic regulation in response to host defenses, linking telomere dynamics to virulence regulation. Experimental disruption of telomerase or telomere-binding proteins often attenuates pathogenicity and reduces stress tolerance, highlighting their functional importance.

Telomere variability and subtelomeric plasticity contribute to the “two-speed genome” phenomenon observed in many fungal pathogens. By compartmentalizing rapidly evolving genes near chromosome ends, fungi exploit telomere dynamics to accelerate adaptation while preserving core essential genes in stable genomic regions. Comparative analyses reveal that telomere sequence motifs and maintenance strategies are diverse across fungal taxa, reflecting adaptation to ecological niches and life history traits.

Correspondence to: Elena Markovic, Department of Fungal Biology and Systems Genomics, University of Belgrade, Belgrade, Serbia, E-mail: e.markovic.mycology@ubgenomics.rs

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CONCLUSION

Telomere maintenance is a central determinant of fungal genome stability, stress tolerance, and evolutionary adaptability. Through telomerase activity, alternative lengthening mechanisms, and subtelomeric regulation, fungi balance

genome integrity with adaptive flexibility. Understanding telomere biology provides insights into fungal evolution, pathogenicity, and potential antifungal targets. Integrating genomic, epigenetic, and functional analyses will continue to elucidate how telomere dynamics shape fungal life history and ecological success.