

# Regulation of the production of fungal cell walls post-transcriptionally

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## ABSTRACT

By coating immunogenic cell wall components like  $\beta$ -glucan with harmless coverings like mannoproteins and  $\alpha$ -glucan that the host is less likely to recognize, pathogenic fungi conceal themselves from their hosts. The success of efforts to comprehend how such processes are controlled has varied. Studies often concentrate on comprehending how fungi respond transcriptionally to either their reservoir environment or the host. However, because of the levels of post-transcriptional and post-translational regulation that take place within a cell, such techniques do not completely address this study subject. Although the effects of post-transcriptional and post-translational regulation have been extensively studied in mammals, we know less about these mechanisms in the kingdom of fungi. Mutations in RNA-binding proteins, such as *Candida albicans* *Slr1* and *Ssd1*, impact the structure of the cell wall and the

pathogenicity of the fungus, demonstrating the importance of post-transcriptional control in these processes. By using research on model yeast and plant pathogenic fungi, we examine the present state of knowledge on fungal post-transcriptional regulation and relate it to putative immune evasion mechanisms. We focus on several RNA-binding proteins that control the production of cell walls and could be involved in the local translation of cell wall constituents. To fully understand fungal virulence mechanisms and for the development of innovative antifungal medicines, it is crucial to increase our understanding of post-transcriptional regulation in human fungal infections.

**Key Words:** *Cardiac rehabilitation; Core components; Guidelines; Heart valve surgery; Heart valve replacement*

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## INTRODUCTION

The multi-layered, active fungal cell wall is made up of proteins and polysaccharides. The inner skeletal layer of chitin and  $\beta$ -glucan form the exoskeleton of the cell and maintains cell shape, rigidity, and turgor pressure. The outer layer of the cell wall is formed of glycosylated mannoproteins, which provide specific cell functions like adhesion and invasion. The composition of the cell wall varies from species to species. When the cell wall is under stress, several transcriptional pathways mount defensive reactions. A core set of cell wall biosynthesis genes are upregulated when the Protein Kinase C (PKC) signaling pathway, which is triggered by the Cell Wall Integrity (CWI) pathway as well as additional pathways like the *HOG1* and calcium-calmodulin pathway, is active through membrane receptors. Histone modifications and transcription factors can control how genes are expressed at the transcriptional level. Non-coding RNAs, upstream open reading frames, RNA binding proteins, mRNA localization factors, and mRNA decay machinery can control gene expression post-transcriptionally. There is a lack of knowledge regarding the regulation of cell wall biosynthesis due to the role of post-transcriptional regulation, despite the wealth of infor-

mation provided by global transcriptional approaches on how genes involved in cell wall biogenesis are differentially regulated in response to various growth and stress conditions.

Alterations to virulence, immunological recognition, and cell wall biosynthesis will occur as a result of the deletion of crucial regulatory units. Deletion of RNA binding proteins involved in the control of cell wall production causes morphological abnormalities, which reduce pathogenicity since cell wall remodeling is necessary for fungal morphogenesis. Aberrant cell wall production can also result in increased PAMP exposure, which can increase Dectin-1 dependent recognition of  $\beta$ -glucan and promote pathogen clearance, similar to what happens when numerous glycosyltransferases are deleted. According to recent advances in crosslinking and proteomic methods, around a sixth of the genome's encoded proteins bind RNA, the majority of which are probably involved in post-transcriptional gene regulation. There is currently little understanding of the functions orthologues of these regulatory proteins perform in pathogenic or filamentous fungus since practically all published information on fungal RNA binding proteins is from *S. cerevisiae*.

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We believe that filamentous fungus will have unique RNA-binding proteins that are lacking from *S. cerevisiae* since they have bigger genomes than yeasts do. Therefore, the significance post-transcriptional regulation plays in cell wall biosynthesis and pathogenicity in fungi is currently greatly underestimated. Genetic screens may be used to find RBPs that have an impact on cell walls, proliferation, and virulence. This paper makes the case that RBPs should be followed up on in similar screening procedures much like transcription factors, protein kinases, and other "usual suspects." RBPs can benefit from in-depth follow-up using molecular genetics, cell biology, structural biology, and other fields of biology.

Characterizing protein interaction partners, in particular, will shed light on the operation and control of RBPs. Beyond looking for symptoms of whole-gene deletions, planned alterations to RNA-binding domains and phosphorylation sites will probably be particularly instructive given that RBPs bind RNA and are frequently controlled by kinases. In conclusion, there is a lot of room for further research into the roles that RNA-binding proteins and localized translation play in fungus. Major findings are probably still to come on the many roles played by post-transcriptional regulation in the regulation of cell walls, environmental responses, and host evasion.