

The 5' untranslated region of the EFG1 transcript promotes its translation to regulate hyphal morphogenesis in *Candida albicans*.

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Abstract

Extensive 5' untranslated regions (UTR) are a hallmark of transcripts determining hyphal morphogenesis in *Candida albicans*. The major transcripts of the EFG1 gene, which are responsible for cellular morphogenesis and metabolism, contain a 5' UTR of up to 1,170 nucleotides (nt). Deletion analyses of the 5' UTR revealed a 218-nt sequence that is required for production of the Efg1 protein and its functions in filamentation, without lowering the level and integrity of the EFG1 transcript. Polysomal analyses revealed that the 218-nt 5' UTR sequence is required for efficient translation of the Efg1 protein. Replacement of the EFG1 open reading frame (ORF) by the heterologous reporter gene CaCBGluc confirmed the positive regulatory importance of the identified 5' UTR sequence. In contrast to other reported transcripts containing extensive 5' UTR sequences, these results indicate the positive translational function of

the 5' UTR sequence in the EFG1 transcript, which is observed in the context of the native EFG1 promoter. It is proposed that the 5' UTR recruits regulatory factors, possibly during emergence of the native transcript, which aid in translation of the EFG1 transcript. **IMPORTANCE** Many of the virulence traits that make *Candida albicans* an important human fungal pathogen are regulated on a transcriptional level. Here, we report an important regulatory contribution of translation, which is exerted by the extensive 5' untranslated regulatory sequence (5' UTR) of the transcript for the protein Efg1, which determines growth, metabolism, and filamentation in the fungus. The presence of the 5' UTR is required for efficient translation of Efg1, to promote filamentation. Because transcripts for many relevant regulators contain extensive 5' UTR sequences, it appears that the virulence of *C. albicans* depends on the combination of transcriptional and translational regulatory mechanisms.

Beteiligte Forschungseinheiten

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