

Factors supporting cysteine tolerance and sulfite production in *Candida albicans*.

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Abstract

The amino acid cysteine has long been known to be toxic at elevated levels for bacteria, fungi, and humans. However, mechanisms of cysteine tolerance in microbes remain largely obscure. Here we show that the human pathogenic yeast *Candida albicans* excretes sulfite when confronted with increasing cysteine concentrations. Mutant construction and phenotypic analysis revealed that sulfite formation from cysteine in *C. albicans* relies on cysteine dioxygenase Cdg1, an enzyme with similar functions in humans. Environmental cysteine induced not only the expression of the CDG1 gene in *C. albicans*, but also the expression of SSU1, encoding a putative sulfite efflux pump. Accordingly, the deletion of SSU1 resulted in enhanced sensitivity of the fungal cells to both cysteine and sulfite. To study the regulation of sulfite/cysteine tolerance in more detail, we screened a *C. albicans* library of transcription factor mutants in the presence of sulfite. This approach and subsequent independent mutant analysis identified the zinc cluster transcription factor Zcf2 to govern sulfite/cysteine tolerance, as well as cysteine-inducible SSU1 and CDG1 gene expression. *cdg1Δ* and *ssu1Δ* mutants displayed reduced hypha formation in the presence

of cysteine, indicating a possible role of the newly proposed mechanisms of cysteine tolerance and sulfite secretion in the pathogenicity of *C. albicans*. Moreover, *cdg1Δ* mutants induced delayed mortality in a mouse model of disseminated infection. Since sulfite is toxic and a potent reducing agent, its production by *C. albicans* suggests diverse roles during host adaptation and pathogenicity.

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