

Csr1/Zap1 maintains zinc homeostasis and influences virulence in *Candida dubliniensis* but is not coupled to morphogenesis.

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[Details](#)



Abstract

The supply and intracellular homeostasis of trace metals are essential for every living organism. Therefore, the struggle for micronutrients between a pathogen and its host is an important determinant in the infection process. In this work, we focus on the acquisition of zinc by *Candida dubliniensis*, an emerging pathogen closely related to *Candida albicans*. We show that the transcription factor Csr1 is essential for *C. dubliniensis* to regulate zinc uptake mechanisms under zinc limitation: it governs the expression of the zinc transporter genes ZRT1, ZRT2, and ZRT3 and of the zincophore gene PRA1. Exclusively, artificial overexpression of ZRT2 partially rescued the growth defect of a *csr1* Δ/Δ mutant in a zinc-restricted environment. Importantly, we found that, in contrast to what is seen in *C. albicans*, Csr1 (also called Zap1) is not a major regulator of

dimorphism in *C. dubliniensis*. However, although a *csr1* Δ/Δ strain showed normal germ tube formation, we detected a clear attenuation in virulence using an embryonated chicken egg infection model. We conclude that, unlike in *C. albicans*, Csr1 seems to be a virulence factor of *C. dubliniensis* that is not coupled to filamentation but is strongly linked to zinc acquisition during pathogenesis.

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Awards

Highlighted as "of special interest" in Ballou ER, Wilson D (2016) Curr Opin Microbiol 32, 128–134.

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