Heavy metal-induced expression of PcaA provides cadmium tolerance to *Aspergillus fumigatus* and supports its virulence in the *Galleria mellonella* model.

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Abstract

Most of the metal transporters in *Aspergillus fumigatus* are yet uncharacterized. Their role in fungal metabolism and virulence remains unclear. This paper describes the novel PIB-type cation ATPase PcaA, which links metal homeostasis and heavy metal tolerance in the opportunistic human pathogen *A. fumigatus*. The protein possesses conserved ATPase motif and shares 51% amino acid sequence identity with the Saccharomyces cerevisiae cadmium exporter Pca1p. A pcaA deletion, an overexpression and a gfp-pcaA complementation strain of *A. fumigatus* were constructed and their heavy metal susceptibilities were studied. The pcaA knock out strain showed drastically decreased cadmium tolerance, however, its growth was not affected by the exposure to high concentrations of copper, iron, zinc, or silver ions. Although the lack of PcaA had no effect on copper adaption, we demonstrated that not only cadmium but also copper ions are able to induce

the transcription of pcaA in *A. fumigatus* wild type Af293. Similarly, cadmium and copper ions could induce the copper exporting ATPase crpA. These data imply a general response on the transcriptomic level to heavy metals in *A. fumigatus* through the induction of detoxification systems. Confocal microscopy of the gfp-pcaA complementation strain expressing functional GFP-PcaA supports the predicted membrane localization of PcaA. The GFP-PcaA fusion protein is located in the plasma membrane of *A. fumigatus* in the presence of cadmium ions. Virulence assays support a function of PcaA for virulence of *A. fumigatus* in the *Galleria mellonella* wax moth larvae model, which might be linked to the elimination of reactive oxygen species.

Involved units

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