The Peroxiredoxin Asp f3 Acts as Redox Sensor in Aspergillus fumigatus.

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Projects

Oxidative inactivation of primary metabolism pathways in the human pathogenic fungus *Aspergillus fumigatus*
Details

Abstract

The human pathogenic fungus Aspergillus fumigatus is readily eradicated by the innate immunity of immunocompetent human hosts, but can cause severe infections, such as invasive aspergillosis (IA), in immunocompromised individuals. During infection, the fungal redox homeostasis can be challenged by reactive oxygen species (ROS), either derived from the oxidative burst of innate immune cells or the action of antifungal drugs. The peroxiredoxin Asp f3 was found to be essential to cause IA in mice, but how Asp f3 integrates with fungal redox homeostasis remains unknown. Here, we show that in vivo, Asp f3 acts as a sensor for ROS. While global transcription in fungal hyphae under minimal growth conditions was fully independent of Asp f3, a robust induction of the oxidative stress response required the presence of the peroxiredoxin. Hyphae devoid of Asp f3 failed to activate several redox active genes, like members of the gliotoxin biosynthesis gene cluster and integral members of the Afyap1 regulon, the central activator of the ROS defense machinery in fungi. Upon deletion of the asp f3 gene Afyap1 displayed significantly reduced nuclear localization during ROS exposure, indicating that Asp f3 can act as an intracellular redox sensor for several target proteins.

Identifier

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