Glucosinolate-derived isothiocyanates impact mitochondrial function in fungal cells and elicit an oxidative stress response necessary for growth recovery

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Titre Glucosinolate-derived isothiocyanates impact mitochondrial function in fungal cells and elicit an oxidative stress response necessary for growth recovery

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Résumé en anglais Glucosinolates are brassicaceous secondary metabolites that have long been considered as chemical shields against pathogen invasion. Isothiocyanates, are glucosinolate-breakdown products that have negative effects on the growth of various fungal species. We explored the mechanism by which isothiocyanates could cause fungal cell death using Alternaria brassicicola, a specialist Brassica pathogens, as model organism. Exposure of the fungus to isothiocyanates led to a decreased oxygen consumption rate, intracellular accumulation of reactive oxygen species and mitochondrial-membrane depolarization. We also found that two major regulators of the response to oxidative stress, i.e. the MAP kinase AbHog1 and the transcription factor AbAP1, were activated in the presence of isothiocyanates. Once activated by isothiocyanate-derived reactive oxygen species, AbAP1 was found to promote the expression of different oxidative-response genes. This response might play a significant role in the protection of the fungus against isothiocyanates as mutants deficient in AbHog1 or AbAP1 were found to be hypersensitive to these metabolites. Moreover, the loss of these genes was accompanied by a significant decrease in aggressiveness on Brassica. We suggest that the robust protection response against isothiocyanate-derived oxidative stress might be a key adaptation mechanism for successful infection of host plants by Brassicaceae-specialist necrotrophs like A. brassicicola.

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