Cell wall integrity and high osmolarity glycerol pathways are required for adaptation of Alternaria brassicicola to cell wall stress caused by brassicaceous indolic phytoalexins

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Résumé en anglais	Camalexin, the characteristic phytoalexin of *Arabidopsis thaliana*, inhibits growth of the fungal necrotroph *Alternaria brassicicola*. This plant metabolite probably exerts its antifungal toxicity by causing cell membrane damage. Here we observed that activation of a cellular response to this damage requires cell wall integrity (CWI) and the high osmolarity glycerol (HOG) pathways. Camalexin was found to activate both AbHog1 and AbSlt2 MAP kinases, and activation of the latter was abrogated in a AbHog1 deficient strain. Mutant strains lacking functional MAP kinases showed hypersensitivity to camalexin and brassinin, a structurally related phytoalexin produced by several cultivated *Brassica* species. Enhanced susceptibility to the membrane permeabilization activity of camalexin was observed for MAP kinase deficient mutants. These results suggest that the two signalling pathways have a pivotal role in regulating a cellular compensatory response to preserve cell integrity during exposure to camalexin. AbHog1 and AbSlt2 deficient mutants had reduced virulence on host plants that may, at least for the latter mutants, partially result from their inability to cope with defence metabolites such as indolic phytoalexins. This constitutes the first evidence that a phytoalexin activates fungal MAP kinases and that outputs of activated cascades contribute to protecting the fungus against antimicrobial plant metabolites.
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