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Clemens, S.; Simm, C.; Maier, T.; Heavy Metal-binding
Proteins and Peptides (2005) DOI: [10.1002/3527600035.bpol8010](https://doi.org/10.1002/3527600035.bpol8010)

Abstract

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IntroductionHistorical OutlineChemical StructuresNomenclature and Structure of
MetallothioneinsPhytochelatin and Phytochelatin-Metal ComplexesStructural Properties of
MetallochaperonesChemical Analysis and
DetectionMetallothioneinsPhytochelatinOccurrenceMetallothioneinsPhytochelatinMetallochaperonesFuncti
onsMetal Homeostasis and the Role of MetallochaperonesBuffering and DetoxificationPhytochelatin
FunctionsMetallothionein FunctionsPhysiologyMetallothionein Localization and IsoformsLocalization and
Compartmentation of Phytochelatin SynthesisBiochemistryMetal-binding Characteristics of
MetallothioneinsBiochemistry of Phytochelatin SynthesisMolecular GeneticsMetallothionein Genes and Their
RegulationPhytochelatin Synthase GenesBiotechnological ApplicationsPatentsOutlook and Perspectives

Scheel, D.; Nuernberger, T.; Signal Transduction in Plant
Defense Responses to Fungal Infection 1-30, (2004)

RIS
BibTeX

0

Rosahl, S.; Feussner, I.; Oxylipins 329-354, (2004)

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RIS
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0

Lee, J.; Nürnberg, T.; Is Pore Formation Activity of HrpZ
Required for Defence Activation in Plant Cells? 165-173, (2003)
DOI: [10.1007/978-94-017-0133-4_18](https://doi.org/10.1007/978-94-017-0133-4_18)

Abstract
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The HrpZ gene product, harpin, is an export substrate of the type III secretion system of phytopathogenic *Pseudomonas syringae*. The role of this protein in pathogenesis is largely unknown. We previously determined that HrpZ binds to lipids and can form cation pores in synthetic lipid bilayers. Such pore-forming activity may allow nutrient release during bacterial colonisation of host plants. In addition, HrpZ is known to trigger plant defence responses in a variety of plants, such as tobacco. We have previously also characterised a binding site in tobacco plasma membranes that likely mediates HrpZ-induced defence responses. In order to reconcile these findings, we pose the question as to whether the activation of plant defence responses by HrpZ is mediated through a “classical” receptor perception mode or if plant membrane perturbation through the inherent pore-forming activity of HrpZ may induce defence responses. As defence in parsley cells can be induced both in a receptor-mediated manner or through ionophores these cells served as an ideal system for our analysis. We first performed ligand binding studies to characterise the presence of a binding site/receptor. We further digested HrpZ with endopeptidases and used subfragments of HrpZ to assess the elicitor-active domain of HrpZ. A C-terminal region of HrpZ appears to be sufficient to elicit plant defence responses. A novel assay involving dye-loaded liposomes was developed to validate previous electrophysiological findings on HrpZ-mediated cation pore formation. More importantly, this assay was used to establish if the elicitor-active C-terminal fragment of HrpZ could form pores. Our findings suggest that the structural requirements for ion pore formation and activation of plant defence responses by HrpZ are different. Thus, ion pore formation alone may not explain the activation of plant defence by HrpZ.

Scheel, D.; Oxidative burst and the role of reactive oxygen
species in plant-pathogen interactions (Inzé, D. & van Montagu,
M., eds.). 137-153, (2002)

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Clemens, S.; Thomine, S.; Schroeder, J. I.; Molecular mechanisms that control plant tolerance to heavy metals and possible roles towards manipulating metal accumulation 665-691, (2002)

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