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Phosphotransfer Protein Ypd1p is Essential for Fludioxonil Action in Phytopathogenic Fungi

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Introduction

Membranes of living cells are permeable to water but not for many dissolved particles. Consequently, cells have to cope with osmotic pressure in the environment. Most of the physiological processes within cells are extremely sensitive towards such changes. Microorganisms, especially fungi, cannot escape unfavourable changing environments, therefore relay on sensitive mechanisms to percept, transform and regulate the responses to these signals by modulating their cellular physiology. In this context, signaling pathways play a crucial role maintaining cellular homeostasis and one example is the high osmolarity glycerol (HOG) signaling pathway responsible for osmoregulation in fungi.

This pathway comprises a sensory phosphorelay system linked to a transferring MAPK cascade, and is well known in the model yeast *Saccharomyces cerevisiae*. In pathogenic fungi the HOG pathway differs accordingly, i.e in the number of histidine kinases in the phosphorelay system, and is yet not well understood. In the research article "High osmolarity glycerol (HOG) signalling in *Magnaporthe oryzae*: Identification of *MoYPD1* and its role in osmoregulation, fungicide action, and pathogenicity" [1], we present the first functional analysis of an *YPD1*-homolog in filamentous phytopathogenic fungi to date. We found that *MoYpd1p* is involved in osmoregulation and pathogenicity. *MoYpd1p* is also involved in the mode of action of the commercial fungicide fludioxonil. Thereby, we improved knowledge about the mechanism of action of fludioxonil by addressing the question, why the group III histidine kinase *MoHik1p* should be the target protein, although directed single gene inactivation of *MoHIK1* is not lethal in *M. oryzae*.

The schemes of the HOG pathway presented in the manuscript display the difference between the modes of action of fludioxonil and single gene inactivation of the gene *MoHIK1*. In case of an inactivation of the target protein *MoHik1p*, the phosphorylation pattern of the phosphorelay system may be maintained by *MoSln1p* and *MoYpd1p* or even by unknown factors, and the mutant strain is viable (Figure 1 left). The proposed mechanism of action of fludioxonil could be assumed to be an interference of fludioxonil with *MoHik1p*, leading to the inhibition of *MoSln1p*.

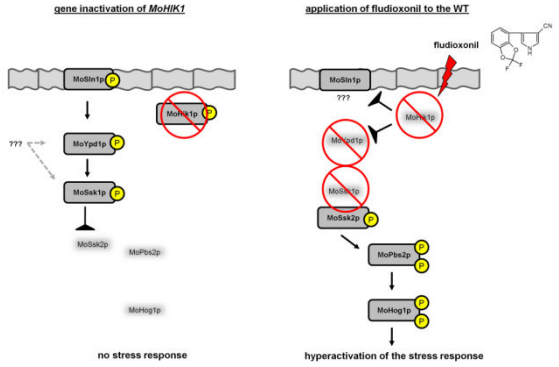

Figure 1: Comparison of the HOG signaling pathway in the *Magnaporthe oryzae* mutant strain *ΔMohik1* (left) and the WT strain (right) upon treatment with fludioxonil. After a directed gene inactivation of *MoHik1p* the phosphorylation pattern of *MoYpd1p* and MoSsk1p can be partially rescued by *MoSln1p*. The response regulator *MoSsk1p* is phosphorylated under normal environmental conditions and the MAPK cascade *MoSsk2p-MoPbs2p-MoHog1p* is inactive. By contrast, fludioxonil treatment of the WT leads to the inhibition of MoHik1p and a subsequent dephosphorylation of all the signaling components of the phosphorelay system resulting in a complete dephosphorylation of *MoSsk1p*. Therefore, the MAPK cascade *MoSsk2p-MoPbs2p-MoHog1p* is hyperactivated, which results in cell death.

Therefore, phopshotransfer via *MoYpd1p* to *MoSsk1p* is inactivated. Consequently, *MoSsk1p* is dephosphorylated and *MoHog1p* hyperactivation causes cell death (Figure 1 right).

References

1. Jacob S, Foster AJ, Yemelin A, Thines E (2015) High osmolarity glycerol (HOG) signaling in Magnaporthe oryzae: Identification of MoYPD1 and its role in osmoregulation, fungicide action and pathogenicity. Fungal Biology 119: 580-594.