Arabidopsis ubiquitin ligase MIEL1 mediates degradation of the transcription factor MYB30 weakening plant defence

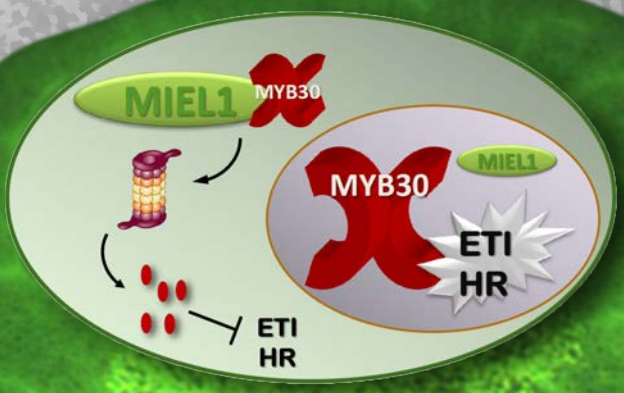
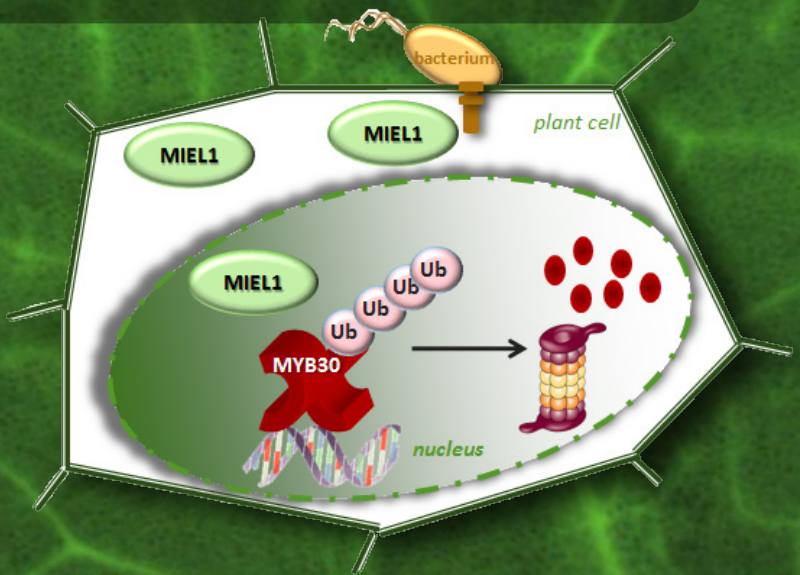


Fig.: Simplified model for the regulation of MYB30-mediated HR cell death through interaction with MIEL1. The action of MIEL1 on MYB30-mediated HR development is presented in cells challenged with bacterial inoculation (brown) and peripheral cells (green). MIEL1 expression is rapidly repressed in challenged cells, indicating that MIEL1 may negatively regulate plant HR and defence activation through degradation of the MYB30 protein in the absence of the pathogen. In addition, repression of MIEL1 in challenged cells may release MYB30 negative regulation, increasing the intensity of the HR and limiting pathogen growth. Finally, MIEL1-mediated degradation of MYB30 may contribute to the spatial restriction of the HR to inoculated cells since MIEL1 expression is not altered in peripheral cells.



One of the most efficient plant resistance reactions to pathogen attack is the hypersensitive response, a form of programmed cell death at infection sites.

The Arabidopsis transcription factor MYB30 is a positive regulator of hypersensitive cell death responses. Here we show that MIEL1 (MYB30-Interacting E3 Ligase1), an Arabidopsis RING-type E3 ubiquitin ligase that interacts with and ubiquitinates MYB30, leads to MYB30 proteasomal degradation and downregulation of its transcriptional activity. In non-infected plants, MIEL1 attenuates cell death and defence through degradation of MYB30. Following bacterial inoculation, repression of MIEL1 expression removes this negative regulation allowing sufficient MYB30 accumulation in the inoculated zone to trigger the hypersensitive response and restrict pathogen growth.

Our work underlines the important role played by ubiquitination to control the hypersensitive response and highlights the sophisticated fine-tuning of plant responses to pathogen attack. Overall, this work emphasizes the importance of protein modification by ubiquitination during the regulation of transcriptional responses to stress in eukaryotic cells.

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