

Roles of Arabidopsis Bax inhibitor-1 in delaying methyl jasmonate-induced leaf senescence

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Previous studies have reported that methyl jasmonate (MeJA) can promote plant senescence. *Arabidopsis thaliana* BI1 (AtBI1) participates in leaf senescence and JA signal pathway. Our recent report has suggested that AtBI1 plays a crucial role in MeJA-induced leaf senescence. Concomitantly, cytosolic calcium $[(Ca^{2+})_{cyt}]$ and MPK6, a mitogen-activated protein kinase (MAPK), participate in the process of MeJA-induced leaf senescence. And *AtBI1* might play its roles in delaying MeJA-induced leaf senescence by suppressing the $[Ca^{2+}]_{cyt}$ -dependent activation of MPK6. Our study contributes to the understanding of the function and mechanism of AtBI1 in plant senescence. Though some of signaling molecules have been elucidated in this type of plant senescence, the mechanism of AtBI1 functions in reducing the $[Ca^{2+}]_{cyt}$ during MeJA-induced leaf senescence needs further improvement, and the source and location of Ca^{2+} are still not clear enough. By using the Arabidopsis and MeJA as the research model, the subsequent researches have been performed to investigate the upstream regulation and downstream function of Ca^{2+} in this type of plant senescence.

Bax inhibitor-1 (BI1) protein was first isolated to prevent Bax-induced cell death in yeast.¹ BI1 has seven trans-membrane domains and predominantly localizes to the ER membrane.² In plants, homologs of BI1 are cloned and characterized to various extents.³ Plant BI-1 plays important roles in the response to many abiotic and biotic stresses. Recent researches have reported

that expression of plant BI1 is upregulated during plant senescence.⁴ Nowadays, more and more attention has been paid to study the mechanism of leaf senescence, which is helpful for understanding of senescence mediation, providing theoretical foundation for screening and genetic manipulation of hyper-resistant crop. Our recent works have shown a function of AtBI-1 during MeJA-induced leaf senescence: overexpression of AtBI-1 delays MeJA-induced leaf senescence (Fig. 1).⁵ We also found that MeJA induced the calcium flux from ER. However, AtBI-1 inhibited the process of calcium flux. Ca^{2+} modulated the activation of MPK6 and then regulated the leaf senescence (Fig. 1).⁵ These findings prompt us to carry out further investigations to determine the signaling mechanism of plant senescence and to dig out the roles of AtBI-1 in plant senescence.

Calcium (Ca^{2+}) is a ubiquitous intracellular second messenger that plays important roles in death pathways.^{6,7} The rise of $[Ca^{2+}]_{cyt}$ is considered as the result of uptake from extracellular or the release from internal stores, such as the endoplasmic reticulum (Fig. 1). It has been reported that Ca^{2+} participates in the regulation of senescence and ripening processes in plants.⁸ Hence, to realize the direct regulatory signal molecules of Ca^{2+} and how does Ca^{2+} translate the senescence signal during the plant senescence will be a meaningful and interesting work, which will provide direct experimental evidences to determine the relationship between AtBI-1 and Ca^{2+} in the senescence network.

Mitogen-activated protein kinase (MAPK) cascades are involved in various

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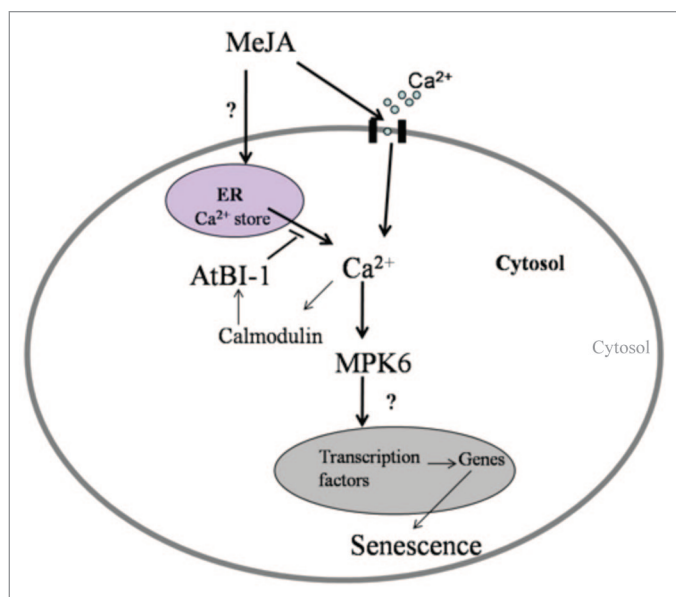


Figure 1. Proposed working model for AtBI-1 delaying MeJA-induced leaf senescence. AtBI1, *Arabidopsis thaliana* Bax inhibitor-1; Ca²⁺, calcium; CaM, calmodulin; MPK6, mitogen-activated protein kinase 6; MeJA, methyl jasmonate. For detailed explanation, see the text.

biotic and abiotic stress responses.^{9,10} It has been suggested that MPK6 plays roles in leaf senescence.⁹ In our paper, the activation of MPK6 was increased during MeJA-induced leaf senescence, and the *mpk6* mutant delayed leaf senescence.⁵ MKK3-MPK6 cascades participate in JA signaling pathway.¹¹ Previous studies suggested that Ca²⁺ might function upstream of the MAPK activation in some stimuli responses. Our data has determined that MeJA-induced MPK6 activation could be arrested after pretreatment with Ca²⁺ scavenger BAPTA-AM.⁵ But it hasn't been known which MAPKK activates MPK6 in the senescence process. It has been reported that a specific MAPK cascade induce the expression of specific WRKY transcription factors.¹³ It is unclear whether MPK6 regulate any WRKY transcription factors

during the MeJA-induced leaf senescence. Our future work will focus on the upstream regulation of Ca²⁺ and downstream function cascades of MPK6 during MeJA-induced leaf senescence. These subsequent researches will certainly contribute to the signal transduction pathways in MeJA-induced leaf senescence.

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