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The ATAF1 transcription factor: At the convergence point of ABA-dependent plant defense against biotic and abiotic stresses

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Because of their sessile lifestyle, plants have evolved sophisticated ways of coping with the various biotic and abiotic stresses they can encounter during their life. Their defensive reactions to a given stress have to be rapid and well adapted to the situation. They are the results of tightly coordinated changes at the molecular level involving the contributions of different signaling pathways. Traditionally, two signal transduction pathways have been investigated preferentially for defense against biotic stresses, attributing a stronger involvement in defense against biotrophic or hemibiotrophic organisms to the salicylic acid (SA)-dependent pathway and a major role in defense against necrotrophs to the jasmonic acid/ethylene (JA/ET)-dependent pathway. In contrast, it is common knowledge that abiotic stress regulation mainly depends on the ABA signaling pathway (for review on the different pathways, see [1]). Recent studies, however, show a role for ABA not only against abiotic stresses but also in defense against pathogens and point to a significant coordination of the plants' responses to the various environmental and biotic stimuli including an important cross-talk between different signaling pathways [2].

NAC (NAM, ATAF, and CUC) transcription factors belong to a gene family specific to plants and play roles in development and stress responses [3]. The first described *NAC* genes *NAM* (***n**o **a**pical **m**eristem*) and *CUC2* (***cu**p-shaped **c**otyledon*), as many later ones, are development-related. In *Arabidopsis* alone, more than a hundred *NAC* genes have been predicted and many of them have overlapping functions. The members of the *ATAF* subfamily of *NAC* domain genes, *ATAF1* and *ATAF2*, were primarily known to negatively regulate responses to drought and wounding and to reduce PR protein expression against some fungi [4, 5], but recent transcription profile data [4] as well as functional studies of *ATAF1* and related *NAC*s show a co-regulated expression by wounding, infection, methyl jasmonate, abscisic acid, hydrogen peroxide, cold, drought, salt and osmotic stresses.

In the non-host plant-pathogen system *Blumeria graminis f. sp. hordei (Bgh) – Arabidopsis*, ATAF1 has been shown to promote penetration resistance [4]. The observed ATAF1-dependent regulation of ABA-responsive genes was clearly correlated to a reduction of ABA levels upon attempted infection of the plants with *Bgh*. In *ataf1* mutants, however, such a *Bgh*-dependent decrease in ABA levels was not observed. Wild-type plants also had higher basal levels of ABA than the mutants (*ataf1*) suggesting that the transcriptional regulation of ABA biosynthesis through ATAF1 is stimulus-dependent. Taken together, this shows that the ability of *Arabidopsis* to restrict penetration by *Bgh* strongly depends on ATAF1-mediated repression of ABA biosynthesis. The obvious hyperinduction, in *Bgh*-infected *ataf1* mutants, of ABA-inducible genes, that normally play a role in abiotic stress resistance, points to a similar antagonistic function of ATAF1 as has been described for ERD15 [6]. Thus, ATAF1 is at the crossroad of biotic and abiotic stress pathways and acts as a switch between plant abiotic stress tolerance and defense.

More, although sometimes controversial, evidence regarding the possible roles of ATAF1 has been published lately. *ataf1* mutants have been described to be more drought tolerant [4, 5] as well as less drought tolerant [7]. In these assays, *ataf1* mutants were grown first on agar medium and then transplanted to soil where water was withheld from them after a certain time of growth. In one case [7] *ataf1* mutants recovered much better than wild type plants after re-watering. On the opposite, and using the same Salk insertional mutants, no differences in the recovery rate were found between both wild type

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and mutants, respectively, by Wu *et al.* [8]. A further controversy can be found after infection with the necrotrophic fungal pathogen *Botrytis cinerea*. Although there is a consensus, that ATAF1-overexpressing lines are more susceptible to *Botrytis* infection [8, 9], the expression patterns of defense genes differed in both cases. While Wu *et al.* [8] describe a down-regulation of *PR1* expression after infection compared to wild type, Wang *et al.* [9] present an up-regulation of *PR1* expression. Similarly, *PDF1.2* expression goes down in one case [8] and remains stable in the other one [9]. Infection with avirulent *Pseudomonas syringae* pv *tomato avr Rpm1* and the virulent wild type *Pst* DC3000 failed to reveal any differences in disease phenotype and severity when comparing wild type and overexpressing lines for *ATAF1* [7] but tests with only *Pst* DC3000 infection of wild type and *ATAF1*-overexpressing lines yielded a higher disease severity in the overexpressing lines when performed in another lab [8].

At this point, it is not possible to resolve where such discrepancies in the results might stem from. The observations are, however, reminiscent of some controversial reports concerning the role of ABA in disease resistance [2, 6]. Lately, evidence has been pointing into the direction of a strong influence of environmental factors in the modulation of the crosstalk between ABA-signaling, and defined biotic and abiotic stress signaling pathways [10]. Any change resulting in an alteration of ABA homeostasis in plants might therefore punctually destabilize the system and alter the balance of the different pathways involved and it looks like ATAF1 might be prominently involved in this process.

In conclusion, there is increasing evidence that transcription factors integrate abiotic stress tolerance and defenses and most reports show that they act through downstream modulation of responses to ABA.

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