SUMMARY

ocp3 mutant plants exhibit an accelerated and intensified callose deposition in response to infection by *Botrytis cinerea* or *Plectosphaerella cucumerina*. After phenotypic analysis of a series of double mutants in *ocp3* background, we observed that both, the increase of callose deposition and resistance, require PMR4 callose synthase and abscisic acid (ABA). In wild plants after infection with any of these necrotrophic fungi, ABA synthesis increases, this increase is even higher in *ocp3*. The greater resistance observed in *ocp3* plants also requires the perception of jasmonic acid (JA) through COI1 receptor. However, JA is not required for the synthesis and basal callose deposition. In this way it could propose a model in which OCP3 exerts a specific control for callose deposition regulated by JA and requires, indispensably, ABA.

The OCP3 protein, by sequence homology, is classified as a nuclear transcription factor family member of Homeobox. However, we found that OCP3 is imported and accumulated in the chloroplasts, and colocalized with pentatricopeptide repeat proteins involved in RNA editing. Specifically, OCP3 participates regulating *ndhB* transcript editing. *ndhB* encodes one subunit of the multiprotein chloroplast complex NADPH dehydrogenase (NDH). The absence of OCP3 results in a decay of *ndhB* editing; a fact which entails a reduction of the activity of NDH complex and therefore leads to a defect in the cyclic electron flow (CEF) around the photosystem I (PSI). We were also able to confirm that mutants having altered activity of NDH complex, as *crr2* and *crr21,* have an increased resistance to infection by *P. cucumerina*, and show an increased callose deposition. Moreover, we note that the editing of the mRNA for other subunits encoded in the chloroplast NDH complex are also regulated, which is significantly altered in the presence of a pathogenic stimuli. At the same time, the stability of NDH complex is also compromised after a pathogenic insult. All these results indicate that the NDH complex is subject to a subtle regulation in accordance with the changing environment.

The ABA is a key player to activate plant defenses against *P. cucumerina* infection. Moreover, hemibiotrof pathogens such as *Pseudomonas syringae* DC3000 also cause increased synthesis of ABA. However, this increase leads to the suppression of defensive responses, which can be attributed a dual role. The regulation of dual role of this hormone, activator or repressor for different defenses, indicates the existence of a post-regulating hormone synthesis. In this Doctoral Thesis, PYR1 has been identified as a family member receptor ABA PYR/PYL/RCAR that perceives this hormone to trigger a defensive response to infection by a pathogen. PYR1 reduces the salicylic acid (SA)-dependent response against the biotrophic pathogens attack. Thus exerts a positive control of JA-dependent responses required to activate the defenses against necrotrophic pathogens. The loss of the perception of ABA through PYR1 activates the expression of SA-responsive genes, modifies the chromatin remodeling and increases the activation of MAP kinases after a biotrophic pathogen attack. Thus, ABA through PYR1 receptor and an epigenetic control, have a regulatory role in plant immunity depending on the lifestyle of pathogens infecting the plant.