Title

Plant hormone-induced defense responses against Botrytis cinerea

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Abstract

Several plant hormones, including ethylene and jasmonates, are known to mediate responses against biotic and abiotic stress. The first and second objectives of this thesis were (i) to elucidate the role of ethylene in defense responses against *Botrytis cinerea* and specifically (ii) to determine the potential contribution of ethylene response factors (ERFs) to induced resistance.

Besides being involved in plant protection, the gaseous plant hormone ethylene is an important developmental signal, controlling fruit ripening and other events. With respect to plant-pathogen interactions ethylene can accelerate or inhibit infection depending on the plant species, organ, or age. Ethylene is known to contribute to foliar resistance against *B. cinerea*. This pathogen causes gray mold and other diseases. It attacks more than 200 plant species, including apple (*Malus domestica*) and pear (*Pyrus communis*). However, effects of ethylene on *B. cinerea* infection of fruits are not well understood. I specifically examined the role of ethylene in defenses of pome fruits against *B. cinerea*. Ethylene was manipulated exogenously and endogenously. Inhibition of ethylene action and biosynthesis was found to stimulate infections of pear and apple fruits by *B. cinerea*, respectively. This implicates ethylene in protecting fruits against *B. cinerea*.

ERFs are known to specifically bind to 'GCC'-boxes of defense-related target gene and to induce foliar resistance against necrotrophic pathogens. To understand the defensive functions of this class of transcription factors in fruits, I characterized members of the *ERF* gene family in apple. Four *MdERF* genes, which are expressed in apple fruits, were identified. To determine dependence of gene expression on ethylene, untransformed and ethylene-silenced apples [Dandekar et al. (2004) Transgenic Res. 13: 373-384] were compared. Only one of them, *MdERF3*, was induced by wounding and *B. cinerea* infection in ethylene-silenced apple fruits. To test the function of MdERF3, the gene was transiently expressed in tobacco leaves. Elevated expression of *MdERF3* was correlated with increased expression of the GCC-box-containing gene *Chitinase 48*. Thus, *MdERF3* appears to be part of the ethylene/pathogenesis-related defense response against *B. cinerea* in apple fruits.

The third objective of my thesis was to investigate interactions between jasmonate and oligogalacturonic acid (OGA) signaling during defense responses against *B. cinerea*. The Cervone hypothesis [Cervone et al. (1989) Plant Physiol. 90: 542-548] states that OGAs generated from *in vitro* interactions between fungal polygalacturonases (PGs) and PG-inhibiting proteins (PGIPs) activate phytoalexin biosynthesis and other plant defense responses. I tested the *in vivo* significance of this hypothesis using genetics. The tomato mutant *coi1* with a defect in the jasmonate signaling was crossed to a transgenic line constitutively expressing PGIP from pear. The results suggest that both jasmonates and PGIP independently alter resistance to this fungal pathogen, thus refuting the hypothesis that OGAs activate the jasmonic acid pathway.