A: CHARACTERIZATION OF PATHOGENS AND PATHOGENESIS

**pthG from Pantoea agglomerans pv. gypsophilae Encodes an Avirulence Effector that Determines Incompatibility in Multiple Beet Species**

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Pantoea agglomerans pv. gypsophilae (Pag) causes root and crown gall disease on gypsophila, whereas *P. agglomerans* pv. beta (Pab) induces the disease on beet as well as gypsophila. Both pathovars harbor a pathogenicity plasmid (pPATHPa9 or pPATHPab) that determines disease development. We have previously isolated and partially characterized a pleiotropic gene from the pPATHPa9 designated as *pthG* that encodes a virulence factor in gypsophila and an elicitor of a hypersensitive-like response in beet roots. The present study was undertaken to characterize *pthG* further as an *avr* gene. Infiltration of beet leaves with strains expressing PthG (i.e., Pag or Pab containing *pthG* in trans) caused an HR response within 48 h, whereas strains lacking intact *pthG* (i.e., Pab or Pag mutated in *pthG*) resulted in gall formation after 5 days. HR was elicited by *pthG* on multiple beet species, whereas a marker exchange mutant of Pag in *pthG* extended its host range on these beet species. A marker exchange mutant of Pag in *hrpJ*, encoding a component of the Type III secretion system, prevented HR elicitation. Mutations in each of the *hrp* regulatory genes (*hrpY, hrpS* and *hrpL*) substantially reduced the transcriptional activity of *pthG* in gypsophila cuttings. Particle bombardment of GFP-PthG fusion caused cell death in beet but not in non-host (melon) leaves. Present and previous results have established *pthG* as a broad-host-range *avr* gene that functions in multiple host plant species and the first functional *avr* gene in *Pantoea* spp. These characteristics of *pthG* may be utilized for creating resistant beet plants against several pathogens. [L]

Anatomical Changes Involved in Gall Formation Caused by Pantoea agglomerans pv. gypsophilae on Gypsophila paniculata

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L = lecture sessions; P = poster (market place) sessions.
Pantoea agglomerans pv. gypsophilae (Pag) is the causal agent of gall formation in Gypsophila paniculata. Its pathogenicity is associated with the presence of an indigenous plasmid (pPATHpa9) that harbors the hrp gene cluster, genes encoding Hop virulence proteins and biosynthetic genes for auxin and cytokinins. Although Type III effectors are crucial for gall initiation by Pag, the hyperplasia and hypertrophy involved in gall formation are triggered by IAA, cytokinin and possibly additional phytohormones. The objectives of the present study were to characterize the patho-anatomy of galls produced by Pag in comparison with tumors induced by Agrobacterium tumefaciens, and in relation to phytohormones synthesis. Microscopic observation of longitudinal section along a gypsophila gall infected with Pag revealed the following patterns: (a) the presence of giant cells surrounded by well developed vascular bundles; (b) formation of circular vessels in parenchyma; (c) suberin deposition on the external surface of the gall cells; and (d) increase in aerenchyma tissue apparently caused by ethylene. Ethylene emission by the wild type, recorded 6 days after inoculation, was eight times higher than by non-infected controls. In contrast, Gypsophila cuttings infected with mutants in the two pathways of IAA biosynthesis and cytokinin showed a significant decline in ethylene production. These mutants also caused limited xylem differentiation and substantial reduction in gall size. Furthermore, giant cells and well developed aerenchyma, which characterize the anatomy patterns of galls induced by the wild type, were absent. The galls produced by Pag were structurally different from tumors caused by A. tumefaciens on Gypsophila: they had a rough appearance, whereas those produced by Agrobacterium were smooth and without suberin. It addition, galls induced by Pag, in contrast to Agrobacterium, had cells with enlarged nuclei containing several nucleoli. Confocal microscopy studies with GFP-labeled Pag showed that the bacteria were located at the edge of the cutting and colonized in small aggregates within the intercellular parenchymal spaces and vessels. Moreover, the presence of the pathogen in planta was essentially limited to the gall area. The bacterial population recorded at 5 cm above the gall was sixfold lower than in the gall tissue.

The Role of Phytohormone in the Interaction between Botrytis cinerea and Arabidopsis thaliana
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The main objectives of the research were to study (a) the impact of phytohormones produced by the plant on the host-parasite interaction by using Arabidopsis thaliana mutants with altered hormone susceptibility or production; and (b) the effect of externally applied phytohormones, inhibitors and precursors on the host-parasite interaction using the plant mutants. Abscisic acid (ABA)-related mutants, both deficient and insensitive, were significantly more susceptible to Botrytis cinerea than the corresponding background lines. External applications of ABA and MVA (mevalonic acid lactone, an ABA precursor) did not change the level of disease on wild type (WT) and the ABA-insensitive mutant abi2-1, but significantly reduced disease on the ABA-deficient mutant aba1-3. Susceptibility of most of the auxin-resistant mutants was similar to that of their WT background; mutants axr1-3 and aux1-7 were more susceptible than WT. There was no influence of NAA (naphthaleneacetic acid) or TIBA (triiodobenzoic acid, an auxin transport inhibitor) on WT, whereas NAA but not TIBA stimulated disease on axr1-3. Ethylene (E)-related mutants included E-insensitive, E-overproducers and E-reduced-production mutants. E-insensitive mutants ein2-1, ein-6, etr1-1 and etr1-3, E-overproducers eto1-1 and eto2, and E-reduced-production mutant his1-1 were more susceptible than WT, whereas other mutants did not differ in susceptibility from their background. Ethephon or AVG (aminoethoxyvinylglycine, an ethylene biosynthesis inhibitor) did not affect the level of disease on WT. AVG significantly inhibited disease on both ein2-1 and his1-1 mutants, whereas ethephon did not affect the level of disease on ein2-1 and slightly stimulated disease.