Title The interaction between ripening tomato fruit and the fungal pathogen Botrytis cinerea

Author Dario Cantu, Ann L. T. Powell, John M. Labavitch, Alan B. Bennett and Eduardo Blumwald

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Abstract

The success of a microbe in colonizing a plant depends on its ability to overcome the constitutive and inducible barriers that the host interposes to limit the invasiveness of a pathogen. Ripening of tomato fruit is associated with increased susceptibility to *Botrytis cinerea* . Histochemical analysis, transcriptome and proteome profiling of infected fruit tissues, together with the phenotype of fruit with altered defense signaling, demonstrate that in response to *B. cinerea* unripe green fruit activate pathogen defense responses that may contribute to unripe fruit resistance. Additionally to defense responses, however, *B. cinerea* induces the expression of genes associated with fruit ripening, including polygalacturonase (*LePG*) and expansin (*LeExp1*), which are among the cell wall proteins that in tomato fruit cooperatively participate in the ripening associated cell wall disassembly. The simultaneous suppression of both LePG and LeExp1 reduces dramatically the ripening associated cell wall disassembly and susceptibility of ripe fruit to *B. cinerea* .

Tomato ripening is regulated independently and cooperatively by ethylene and transcription factors, including *NOR* and *RIN* . Mutations in *NOR* or *RIN* or interference with ethylene perception prevent fruit from ripening, whereas only a mutation in *NOR* renders fruit resistant to *B. cinerea* , suggesting that not all ripening pathways render fruit susceptible. Significantly, *LePG* and *LeExp1* are only induced in susceptible *rin* fruit and not in resistant *nor* fruit. That the fungal secretome does not change regardless of fruit ripening further supports the dependence of fungal virulence on fruit functions.

These results demonstrate that *B. cinerea* virulence depends on some, but not all ripening processes. The onset of ripening marks the completion of seed maturation when fruit become agents of seed dispersal, a process that may be facilitated by ripening associated pathogen susceptibility. The co-evolution of fruit with their pathogens may be responsible for the relationship between ripening and pathogen susceptibility of ripe fruit. The demonstrated pathogen dependence on the self-disassembly of ripening fruit cell walls assures that seeds mature before fruit decomposition is promoted. Thus, the

activity of fungal virulence factors and fruit ripening functions combine to contribute to the completion of the life cycles of both plant and pathogen