Title Fruit exocarp phenols in relation to quiescence and development of *Monilinia fructicola* Infections in *Prunus* spp.: a role for cellular redox?

Authors Miin-Huey Lee and Richard M. Bostock

Citation Phytopathology 97 (3): 269-277. 2007.

Keywords disease resistance; latent infection; postharvest diseases.

Abstract

Monilinia fructicola causes brown rot of *Prunus* species and usually remains quiescent on immature fruit but reactivates when fruit are mature. The dihydroxycinnamates caffeic acid and its quinate ester, chlorogenic acid, abundant in the exocarp of peach fruit, had no effect on fungal growth but markedly inhibited the production of the cell wall degrading enzymes polygalacturonase and cutinase in *M. fructicola* cultures. This inhibition was related to changes in the electrochemical redox potentials of the cultures, as measured with a redox electrode. Fungal culture filtrates had lower electrochemical redox potentials when the growth medium contained caffeic acid than in caffeic acid-free medium. Levels of total intracellular glutathione, the reduced form of which serves as a major cellular antioxidant, increased significantly in *M. fructicola* cells in response to external caffeic acid. The presence of caffeic acid, chlorogenic acid, or reduced glutathione in conidial suspensions of *M. fructicola* did not inhibit germination on flower petals and fruit, but inhibited appressorium formation from germinated conidia and subsequent brown rot lesion development. These results suggest that intracellular antioxidant levels in the pathogen can be influenced by phenols present in host tissue and that changes in the redox environment may influence gene expression and differentiation of structures associated with infection by the pathogen. The possible relationship of host phenols to quiescence and subsequent development of *M. fructicola* infections is discussed.