Title Induction, regulation, and role in pathogenesis of appressoria in *Monilinia fructicola* infections in *Prunus* spp.: A role for cellular redox?

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Abstract

Monilinia fructicola, which causes brown rot in stone fruit, forms appressoria on plant and artificial surfaces. On nectarine, the frequency of appressoria produced by conidial germlings depends to a large degree on the stage of fruit development, with numerous appressoria formed on immature (stage II) nectarine fruit, and no appressoria observed on fully mature fruit (late stage III). On polystyrene surfaces, appressorium formation was increased from <10% of germinated conidia to >95% of germinated conidia when the conidia were washed to remove residual nutrients and self-inhibitors. *M. fructicola* appressorium formation also appears to be regulated by the topography of the plant surface. On fruit, appressoria formed on stomatal guard cell lips, on the grooves of lateral cells adjacent to stomata or between two epidermal cells, and on the convex surfaces of epidermal cells. Pharmacological effectors indicate that cyclic AMP-, MAP kinase-, and calcium/calmodulin-dependent signaling pathways are involved in the induction and development of appressoria. KN-93, an inhibitor of calmodulin-dependent protein kinase II, did not inhibit conidial germination but did inhibit appressorium formation and brown rot development on flower petals, suggesting that appressoria are required for full symptom development on *Prunus* spp. petals.