The Important Genes for Infection of *Colletotrichum spp.*

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Abstract

Colletotrichum spp. is an important fungal pathogen for many crops, i.e. *C. lagenarium*. In the infection process, *Colletotrichum* species need to penetrate the outer layers of plant in order to colonize host tissues. The process includes three stages: conidial germination, appressorium differentiation and development, and penetration peg formation for entry into hosts. The initial stage of infection is the triggering of conidium germination and appressorium formation by the surface compounds of the hosts. It has been found that a protein kinase (also known as *LIPK* (lipid-induced protein kinase)) from C. *trifolii* can be induced by lipids. The results from the genetic disruption experiments showed that *LIPK* related to appressorium differentiation [(7)]. Furthermore, mitogen-activated protein kinase (MAP kinase) regulated diverse steps in the infection process including conidial germination and appressorium differentiation. In the *C. lagenarium*, two MAP kinases (*Maf1* and *Cmk1*) have been reported to be involved in fungal pathogenicity [(12, 19)]. There was a significant reduction in fungal pathogenicity for *Maf1* gene replacement mutants. The *Maf1* mutants germinated normally, but failed to form appressorium. *Cmk1* mutants revealed that *Cmk1* regulated *Colletotrichum* conidial germination, appressorium differentiation, and subsequent invasive growth in host plants. The results showed that *Cmk1* mutants failed to germinate. However, the addition of yeast extract rescued germination, indicating the presence of an unknown mechanism for regulation of conidial germination. Yamauchi et al. (2004) reported that the adenylate cyclase gene (*Cac1*) and cyclic AMP (cAMP) regulated the signal transduction pathway of protein kinase A (PKA) and then influenced the conidial germination of *Colletotrichum* spp. The maturing *Colletotrium* still needs to synthesize melanins and forms penetration pegs after appressorium formation. The *Cst1* gene from *C.*

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lagenarium and the *CIPLS1* gene from *C. lindemuthianum* regulated the formation of the penetration peg [20, 21]. *Cst1* and *CIPLS1* disruption mutants were able to produce the appressorium with melanins, but could not infect the host surface normally. According to these observations, we understand that a successful infection of *Colletotrium* not only requires the formation of the melanined appressorium for generating turgor pressure, but also the factors for cytoskeleton regulation.

Key words：*Colletotrichum spp*, MAP kinase, Appressorium, Penetration peg.