Sclerotinia sclerotiorum is a necrotrophic fungal pathogen causing white mold disease on more than 600 plant species, including many economically important crops (1,2,3). The most prominent symptom of white mold is maceration of host tissue, suggesting the efficiency of pathogen in degrading plant cell wall (3,4). Galacturonic acid is the major building block of pectin which is a main component of plant cell wall. Thus, the resulting galacturonic acid after tissue maceration is likely the nutrient source for S. sclerotiorum. The genome of S. sclerotiorum encodes the genes responsible for galacturonic acid catabolism (5,6). However, the roles of these galacturonic acid catabolic genes in the biology and virulence of S. sclerotiorum are unknown.

The D-galacturonic acid catabolic pathway in S. sclerotiorum consists of three catalytic steps converting D-galacturonic acid to pyruvate and L-glyceraldehyde (7). In an effort to characterize the functions of the galacturonic acid catabolic pathway genes, gene deletion mutants of these genes in S. sclerotiorum were generated using targeted mutagenesis.

The wildtype and gene-deletion mutant strains of S. sclerotiorum were tested on media with different carbon sources. For radial growth assays, mycelium of strains were inoculated on Murashige and Skoog media supplemented with Ammonium dihydrogen phosphate and as a carbon source either glucose, D-galacturonic acid, citrus fruit pectin, apple pectin or sodium pectate. The significance of the effects of Sclerotinia sclerotiorum and the characterization of its virulence in host plants will be discussed in efforts to understand the epidemiology of the disease.