MODIFICATION OF PHYTOALEXIN STRUCTURE TO IMPROVE COTTON RESISTANCE TO WILTS

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Abstract

For a host-pathogen incompatible response (i.e., plant exhibits resistance), the plant must recognize the pathogen quickly and then rapidly begin the process of marshalling its defenses. Recent experiments from our laboratories shows that in new Verticillium wilt resistant cottons, mRNA levels for genes involved in lignin and phytoalexin synthesis reach maximal activity 12 hours after inoculation with conidia of *Verticillium dahliae* (defoliating isolate V-76). Since the new resistant cultivars already exhibit early recognition of the pathogen, it behooves us to focus on improving other defense mechanisms in order to further increase resistance. Numerous studies from our laboratories have demonstrated that the rapid biosynthesis of the phytoalexins play an essential role in cotton's active defense response. Because of the phytoalexin's critical role in resistance, we have focused our research on enhancing the potency of these compounds via two routes: 1) alter the current cotton phytoalexin composition and 2) introduce genes from related genera that will yield different more toxic phytoalexins.

Cotton synthesizes four phytoalexins in response to infection by *V. dahliae*: desoxyhemigossypol (dHG), hemigossypol (HG) and their methyl ether derivatives desoxyhemigossypol-6-methyl ether (dMHG) and hemigossypol-6-methyl ether (MHG). Bioassays show that the methyl ether derivatives are approximately one-half as toxic to several soilborne pathogens (i.e., *V. dahliae*, *Fusarium oxysporum*, f. sp. *vasinfectum*, and *Rhizoctonia solani*) compared to their unmethylated parent compounds. We have also shown that dHG acts as the unique substrate for dHG-O-methyltransferase (dHG-OMT) producing dMHG. MHG is produced by oxidation of dMHG, not by O-methylation of HG. Our immediate goal is to isolate dHG-OMT, sequence the enzyme, clone the gene, express it in *Escherichia coli*, prepare antisense constructs, and express these in cotton. Thus, we expect to significantly reduce the degree of methylation in the cotton phytoalexins and enhance the toxicity of the phytoalexins produced by cotton.

Kenaf (*Hibiscus cannabinus*) is very resistant to *V. dahliae*, and it produces a phytoalexin [*o*-hibiscanone (HBQ)] that is ~ 8 times as toxic to this pathogen as the most potent phytoalexin in cotton. HBQ appears to be biosynthesized *via* the same route as the cotton phytoalexins. Thus, it appears that the introduction and expression of only one to three genes from kenaf into cotton would significantly alter the plant's secondary product biochemistry and substantially increase pathogen resistance.

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