

Characterization of the dry bean polygalacturonase-inhibiting protein (PGIP) gene family during *Sclerotinia sclerotiorum* (Sclerotiniaceae) infection

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ABSTRACT. Polygalacturonase-inhibiting proteins are leucine-rich repeat proteins that inhibit fungal endopolygalacturonases. The interaction of polygalacturonase-inhibiting protein with endopolygalacturonases limits the destructive potential of endopolygalacturonases and may trigger plant defense responses induced by oligogalacturonides. We examined the expression of fungal pg and plant Pvpgip genes in bean (Phaseolus vulgaris) stems infected with Sclerotinia sclerotiorum to determine whether any of them are associated with the infection process. Transcriptional analysis was carried out by means of semi-quantitative reverse transcription PCR or real-time PCR. The sspg1 gene was highly expressed during infection; sspg3 was regulated during the later phases of infection; sspg5 was more uniformly expressed during infection, whereas *sspg6* was only weakly expressed. During the course of infection, *Pvpgip1* transcripts were not detected at early stages, but they appeared 72 h post-inoculation. High levels of Pvpgip2 expression were observed during the initial phase of infection; the transcript peaked by 48 h post-inoculation and declined by 72 h post-inoculation. *Pvpgip3* expression increased strongly at 96 h post-inoculation. *Pvpgip4* was constantly present from 24 h post-inoculation until the end of the experiment. However, we detected higher levels of the *Pvpgip4* transcript in the necrotic lesion area than in plants that had been mechanically wounded. Remarkably, only *Pvpgip4* appeared to be moderately induced by mechanical wounding. These results provide evidence that endopolygalacturonases contribute to the infection process during host colonization by promoting the release of plant cell oligogalacturonides, which are powerful signaling molecules and may also activate plant defenses, such as polygalacturonase-inhibiting proteins.

Key words: Endopolygalacturonase; *Phaseolus vulgaris*; White mold; Polygalacturonase-inhibiting protein; *Sclerotinia sclerotiorum*