

FUNGAL PATHOGENS CAN ELUDE THE INHIBITION BY HOST PLANT POLYGALACTURONASE INHIBITING PROTEINS (PGIPs) BY DIFFERENT MECHANISMS

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To infect and colonize successfully host plants, fungal pathogens have evolved specific strategies. Many fungi overcome the defensive barrier represented by the cell wall by producing large amount of cell wall degrading enzymes (CWDE) during infection. Among CWDE, pectinases, and especially endo-polygalacturonases (endo-PG), play an important role since they depolymerise the pectic component of the cell wall and middle lamella. Polygalacturonase-inhibiting proteins (PGIPs) are plant defence molecules able to block the polygalacturonase activity of fungal pathogens. Therefore, PGIP could be used both in traditional and innovative breeding programmes to defeat those pathogens that express endo-PG activity to assist with plant infection. However, fungi have evolved strategies to overcome PGIP inhibition. For example, *Sclerotinia sclerotiorum* in the early stage of soybean infection produces considerable amount of a basic PG encoded by the *Sspgb* gene. By quantitative RT-PCR it was shown that, initially, the transcript of this *pg* gene largely overcomes that of the soybean *pgip* gene. Therefore, at this stage, the PGIP level would be not enough to significantly counteract fungal PG activity.

Later, the level of the *pgip* transcript increases in infected tissue but, at the same time, the fungus accumu-

lates a second *pg* gene (*Sspga*) coding for an acidic PG. This PG is less sensitive to PGIP inhibition under the acidic pH conditions determined in diseased tissue by fungal secretion of oxalic acid. In other cases, PGIPs may fail to recognise specific PGs produced by some fungi. For example, a PG refractory to all host and non-host PGIPs so far characterized was isolated from a *Fusarium moniliforme* (syn. *F. verticillioides*) strain, and the *pg* gene encoding this PG was cloned. The comparison of the amino acidic sequence of this PG with that obtained from a different *F. moniliforme* isolate, susceptible to bean and soybean PGIP inhibition, allowed the identification of a few amino acidic substitutions putatively responsible for escaping inhibition. Further studies using overlap extension and site-directed mutagenesis will allow the identification of the specific amino acids responsible for the lack of PG-PGIP recognition. Therefore, although over expression of *pgip* genes could be considered as a possible strategy to increase plant resistance, the effectiveness of plant PGIPs may depend on the specific molecular characteristics of fungal PGs, and also on the presence of additional factors such as oxalic acid that can influence PG activity directly or indirectly, by favouring its escape from PGIP inhibition.

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