REGULATION AND ROLE OFPECTINASES IN PHYTOPATHOGENIC Fungi

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Abstract

Fungal plant pathogens secrete a large array of cell wall hydrolases. Among them, pectinases play a dual role during the interaction between the fungus and the host plant. Pectin degradation contributes to fungal virulence but also induces defence gene expression in the host plant. To evaluate the role of pectinases in these different aspects of the interaction between plants and filamentous fungi, strategies involving gene disruption, reporter genes and site-directed mutagenesis are being used. In this chapter, we will review recent studies aiming to analyse the role of pectinases in the outcome of different plant-fungus interactions.

1. Introduction

The plant cell wall is a potential barrier to the penetration and spread of fungal pathogens in plant tissues and most of plant parasites produce enzymes that can degrade cell wall polymers. Pectinases, including pectin and pectate lyases, polygalacturonases, pectin-methyl esterases and rhamnogalacturonases, are the first cell wall-degrading enzymes produced during infections and are thought to play a major role in pathogenesis. Pectinases are encoded by gene families whose size appears to vary with the type of infection. For instance, necrotrophic pathogens such as Botrytis cinerea and Sclerotinia sclerotiorum which attack a large number of plant species produced a large set of endopolygalacturonase (endoPG) isoforms (Fraissinet-Tachet et al., 1995; Wubben et al., 1999) whereas biotrophic and hemibiotrophic pathogens with a restricted host range such as Colletotrichum lindemuthianum and Claviceps purpurea contain only two different endoPG genes (Centis et al., 1996; Centis et al., 1997; Tenberge et al., 1996). On the other hand, pectinases appear to induce very efficiently plant defense reactions probably through the release of pectic fragments which could act as endogenous elicitors. In this article, we will review recent progress made in the understanding of the regulation of pectinase production in phytopathogenic fungi, and the role of these enzymes during the interaction with the host plant.
2. Role of pectinases in pathogenesis

Gene disruption experiments of pectinase genes were performed in several phytopathogenic fungi (Table 1). Most of the mutant strains retain their pathogenicity (Gao et al., 1996; Garcia-Maceira et al., 2001; Scott-Craig et al., 1998; Scott-Craig et al., 1990) but a clear involvement of pectinase genes has been established in macerating fungi such as Alternaria citri (Isshiki et al., 2001) and B. cinerea (ten Have et al., 1998). From these different studies it can be concluded that the importance of pectinolytic enzymes in pathogenicity will vary with the type of disease and the host plant. However, it must be pointed out that residual pectinolytic activities were detected in the disrupted strains suggesting that functional pectinase genes were still present.

Actually, gene redundancy is a major obstacle to determine the importance of pectinolytic activity in pathogenesis. To circumvent this problem, all the genes coding a given enzymatic activity could be disrupted. Recently, two pectate lyase genes were inactivated in the pea pathogen Nectria hematococca (Rogers et al., 2000) leading to a strong reduction of virulence. However, this strategy is feasible only in the case of a small number of genes. An other approach is to inactivate genes involved in the transcriptional induction of pectinases genes. This strategy was successful in the case of the maize pathogen Cochliobolus carbonum. The gene ccSFNI, an ortholog of the yeast regulator SFNI, was shown to be essential for the expression of a large set of cell-wall degrading enzymes and for pathogenesis (Tonukari et al., 2000). However, ccSFNI is a general transcriptional regulator for glucose-repressed genes, and mutations in ccSFNI can affect expression of unknown genes specifically involved in pathogenesis. Thus, isolation of regulators specific for pectin-induced genes is necessary to investigate the role of pectinases in pathogenesis.

Finally, expression of a new pectinase in a pathogenic fungus can be sufficient to promote pathogenicity. This was shown in the case of Colletotrichum magna, a pathogen of cucurbit, which was transformed with a pectate lyase gene from the avocado pathogen Colletotrichum gloeosporioides. Transformed isolates became more aggressive on watermelon and avocado (Yakoby et al., 2000). However, expression of an endoPG gene from Fusarium oxysporum f.sp. lycopersici in deficient strains of F. oxysporum f.sp. melonis did not induce any change in pathogenicity (Di Pietro and Roncero, 1998).