Phytochemistry xxx (2014) xxx–xxx

Contents lists available at ScienceDirect

Phytochemistry

journal homepage: www.elsevier.com/locate/phytochem

Review

Interplays between the cell wall and phytohormones in interaction between plants and necrotrophic pathogens

Majse Nafisi, Lorenzo Fimognari, Yumiko Sakuragi *

Department of Plant and Environmental Sciences, University of Copenhagen, Thorvaldsensvej 40, Frederiksberg 1871, Denmark

ARTICLE INFO

Article history:
Available online xxxx

This paper forms part of a special issue of Phytochemistry dedicated to the memory and legacy of Professor (Godfrey) Paul Bolwell, MA DSc (Oxon). (1946–2012), internationally-recognised plant biochemist and Regional Editor of Phytochemistry (2004–2012). He is much missed by his friends.

Keywords:
Plant cell wall
Necrotrophic phytopathogens
Disease resistance
Auxin
Cytokinin
Brassinosteroids
Abscisic acid

ABSTRACT

The plant cell wall surrounds every cell in plants. During microbial infection, the cell wall provides a dynamic interface for interaction with necrotrophic phytopathogens as a rich source of carbohydrates for the growth of pathogens, as a physical barrier restricting the progression of the pathogens, and as an integrity sensory system that can activate intracellular signaling cascades and ultimately lead to a multitude of inducible host defense responses. Studies over the last decade have provided evidence of interplays between the cell wall and phytohormone signaling. This review summarizes the current state of knowledge about the cell wall-phytohormone interplays, with the focus on auxin, cytokinin, brassinosteroids, and abscisic acid, and discuss how they impact the outcome of plant–necrotrophic pathogen interaction.

Contents

* Corresponding author. Tel.: +45 35333317.
E-mail address: ysa@plen.ku.dk (Y. Sakuragi).

http://dx.doi.org/10.1016/j.phytochem.2014.11.008
0031-9422/

1. Introduction

The plant cell wall surrounds every cell in plants and forms a dynamic physical barrier that protects the cell from microbial infection. The main constituents of the plant cell wall are cellulose, hemicelluloses, pectins, and glycoproteins (Carpita and Gibeaut, 1993). Cellulose form crystalline microfibers and provides a scaffold to the cell wall, hemicelluloses crosslink with celluloses to provide support to the cellulose microfibrils network, while pectins not only crosslink cell wall polymers but also serve as hydrated extracellular matrix components (Somerville et al., 2004). The exact composition of the cell wall polysaccharides varies between tissues and can differ in the same tissues during developmental processes. During pathogen infections, the cell wall undergoes dramatic structural and chemical changes (Eggert et al., 2014; Voigt, 2014; Vorwerk et al., 2004), including lignification (Vance et al., 1980; Zhao and Dixon, 2014), deposition of callose (Luna et al., 2011), cell wall protein cross-linking (Bradley et al., 1992), accumulation of reactive oxygen species and antimicrobial compounds (phytoalexins) (Franke et al., 2005; Lamb and Dixon, 1997; O'Brien et al., 2012), which can culminate to restrict the infection and prevent further pathogen progression. Emerging notion is that the cell wall integrity is sensed by plants and, when compromised, it can activate intracellular events involving phytohormone signaling cascades that can in turn activate defense responses (Hamann, 2012). In addition, degradation of cell wall constituents, particularly by necrotrophic pathogens, are sensed by a plasmamembrane receptor(s), leading to activation of defense signaling cascades and eventual mounting of inducible defense responses (Fry et al., 1993; Monaghan and Zipfel, 2012).

The aim of the present review is to provide a brief summary of the current state of our understanding regarding the interplay between plant cell wall integrity and phytohormones in the context of defense against necrotrophic pathogens. Plant pathogens are often classified into two major classes: necrotrophic and biotrophic pathogens. Lifestyle, infection strategy, and host defense responses vary greatly between the two classes. Necrotrophic pathogens use a suite of cell wall degrading enzymes and toxins to kill and macerate the host tissues to feed on; in contrast, biotrophic pathogens cause relatively minor damage on the host cell wall and maintain host viability to acquire nutrients. Some plant pathogens can manifest biotrophic-like strategies at earlier stages of infection and then switch to necrotrophic-like strategies at later stages; such pathogens are referred to as hemibiotrophic (Laluk and Mengiste, 2010). The cell wall maceration and damages on the host tissue by necrotrophic phytopathogens causes devastating economic losses in agriculture (Williamson et al., 2007). In the *Botrytis cinerea* genome, the large capacity for plant cell wall degradation was illustrated by the identification of 118 genes unambiguously associated with plant cell wall degradation (Amselem et al., 2011). Plants defend against necrotrophic phytopathogens through a complex interplay of phytohormone signaling and defense responses, and this topic has been extensively reviewed during the past few years (Cao et al., 2011; De Bruyne et al., 2014; De Vleesschauwer et al., 2013; Grant and Jones, 2009; Perez and Goossens, 2013; Pieterse et al., 2012). Thus plant–necrotrophic pathogens interaction offers unique and valuable insights into interplays between cell wall stress perception and phytohormone signaling cascades that culminate to determine the necrotrophic disease outcome. In this review we discuss (i) roles of phytohormones, namely auxin, cytokinin, brassinosteroids (BR), and abscisic acid (ABA), in defense against necrotrophic phytopathogens, (ii) how these phytohormones modulate the cell wall properties, and (iii) how the cell wall can modulate the homeostasis of these phytohormones signalings and impact pathogen resistance. Although the main focus is on the interaction between the plant cell wall and necrotrophic pathogens, evidence based on interaction between plants and biotrophic pathogens are discussed where relevant. Involvement of jasmonates (JA), ethylene (ET), and salicylic acid (SA) in the cell wall-mediated defense has been extensively discussed in recent years (Hamann, 2012; Malinovsky et al., 2014) and will not be dealt with in this article.

2. Auxin

2.1. Roles of auxin in plant defense against necrotrophic pathogens

In plants, auxin can be found in the forms indole-3-acetic acid (IAA), 4-chloroindole-3-acetic acid, phenylacetic acid, and indole-3-butyric acid, however, IAA is the most potent auxin in plants (Woodward and Bartel, 2005). The negative impact of auxin in SA-mediated defense against biotrophic pathogens has recently been demonstrated (Chen et al., 2007; Wang et al., 2007) and a role(s) of auxin in defense against necrotrophic pathogens is also emerging (Kazan and Manners, 2009; Korolev et al., 2008; Llorente et al., 2008). Treatment of plants with the auxin transport inhibitor, 2,3,5-triiodobenzoic acid, has been shown to lead to increased necrotrophic infection by *Plectosphaerella cucumerina* (Llorente et al., 2008). Furthermore, the *aux1* mutant, which is defective in auxin influx, cannot develop induced systemic resistance against *Botrytis cinerea* (Korolev et al., 2008) and auxin signaling mutants, *axr1*, *axr2* and *axr6,* are more susceptible towards *B. cinerea* and *P. cucumerina* than the wild type (Korolev et al., 2008; Llorente et al., 2008). The altered defense response of auxin signaling mutants towards *B. cinerea* and *P. cucumerina* does not appear to be due to altered activation of SA and JA/ethylene defensive pathways as the expression of marker genes *PR1* and *PDF1.2* was not altered. These data suggest that auxin signaling is required for full defense capacity towards *B. cinerea* and *P. cucumerina* in Arabidopsis. The authors speculated that the changes in disease outcome in response to exogenous IAA were not directly caused by the hormone because IAA can induce ethylene and the IAA-induced resistance was not observed in the ethylene insensitive *ein2* mutant (Savatin et al., 2011). In the case of enhanced susceptibility, it was suggested that conversion of IAA to IAA-Asp is responsible (González-Lamothe et al., 2012). The study showed that the infection by *B. cinerea* increases the level of IAA-Asp in Arabidopsis and exogenous application of IAA-Asp and IAA, but not the synthetic auxin, 2,4D, enhanced susceptibility to *B. cinerea* and *Pseudomonas syringae*. Conversely the *gh3.2* mutant, defective in an auxin-conjugating enzyme and as a result has a lower level of IAA-Asp, exhibited enhanced resistance to *B. cinerea* and *P. syringae*. It was found that IAA-Asp promotes the expression of certain virulence genes in the pathogens during infection of plants (González-Lamothe et al., 2012).

It is noteworthy that *B. cinerea* can synthesize auxin and secrete it to the media when grown *in vitro* (Sharon et al., 2007), but the exact function of the pathogen-derived IAA during infection and in interaction with the plant host has not been elucidated. Following infection of Arabidopsis by *B. cinerea*, a number of the host IAA

Please cite this article in press as: Nafisi, M., et al. Interplays between the cell wall and phytohormones in interaction between plants and necrotrophic pathogens. Phytochemistry (2014), http://dx.doi.org/10.1016/j.phytochem.2014.11.008