Phytochemistry xxx (2014) xxx–xxx

Contents lists available at ScienceDirect

Phytochemistry

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

Review

Roles of apoplastic peroxidases in plant response to wounding

Farida Minibayeva [a,*], Richard Peter Beckett [b], Ilse Kranner [c]

[a] Kazan Institute of Biochemistry and Biophysics, Russian Academy of Sciences, P.O. Box 30, Kazan 420111, Russian Federation
[b] School of Life Sciences, PBag X01, Scottsville 3209, University of KwaZulu-Natal, Pietermaritzburg, South Africa
[c] Institute of Botany, University of Innsbruck, Sternwartestraße 15, A-6020 Innsbruck, Austria

ARTICLE INFO

Article history:
Available online xxxx

Keywords:
Plant
Wounding stress
Peroxidases
Reactive oxygen species
Apoplast
Wound healing
Cell death

ABSTRACT

Apoplastic class III peroxidases (EC 1.11.1.7) play key roles in the response of plants to pathogen infection and abiotic stresses, including wounding. Wounding is a common stress for plants that can be caused by insect or animal grazing or trampling, or result from agricultural practices. Typically, mechanical damage to a plant immediately induces a rapid release and activation of apoplastic peroxidases, and an oxidative burst of reactive oxygen species (ROS), followed by the upregulation of peroxidase genes. We discuss how plants control the expression of peroxidases genes upon wounding, and also the sparse information on peroxidase-mediated signal transduction pathways. Evidence reviewed here suggests that in many plants production of the ROS that comprise the initial oxidative burst results from a complex interplay of peroxidases with other apoplastic enzymes. Later responses following wounding include various forms of tissue healing, for example through peroxidase-dependent suberinization, or cell death. Limited data suggest that ROS-mediated death signalling during the wound response may involve the peroxidase network, together with other redox molecules. In conclusion, the ability of peroxidases to both generate and scavenge ROS plays a key role in the involvement of these enigmatic enzymes in plant stress tolerance.

Contents

1. Introduction

Plants frequently suffer from wounding, for example by insect or animal grazing or trampling, or agricultural practices, and display an array of responses to wounding. Wounding destroys cells and tissues, making organisms vulnerable to pathogen infection. In animals, various cell types including white blood cells, platelets, epithelial cells and vascular smooth muscle cells cooperate in response to wounding (Sen and Roy, 2008). Multiple processes including cell migration and proliferation, and the differentiation and synthesis of extracellular matrix components are initiated after injury to repair epithelial tissues and blood vessels and to kill bacteria around wound sites. Plants do not possess mechanisms to mobilize cells in response to wounding, because plant cells are surrounded by rigid cell walls. Plant wound responses

Abbreviations: ABA, abscisic acid; DPI, diphenylene iodonium; JA, jasmonic acid; MJ, methyl jasmonate; PCD, programmed cell death; ROS, reactive oxygen species; Rboh, respiratory burst oxidase protein; SA, salicylic acid.

* Corresponding author. Tel.: +7 (843) 2319045; fax: +7 (843) 2927347.
E-mail addresses: minibayeva@mail.knc.ru, fminibayeva@gmail.com (F. Minibayeva), rpbeckett@gmail.com (R.P. Beckett), Ilse.Kranner@uibk.ac.at (I. Kranner).

http://dx.doi.org/10.1016/j.phytochem.2014.06.008
0031-9422/

involve a transient production of reactive oxygen species (ROS), the "oxidative burst" (Minibayeva et al., 1998; Orozco-Cárdenas et al., 2001; Ross et al., 2006), an activation of the phenylpropanoid pathway (Collinge and Slusarenko, 1987), deposition of callose and polymerized phenolics, cell wall cross-linking (Bradley et al., 1992), cell wall suberinization (Bernards et al., 1999), and controlled cell death (Cui et al., 2013). The production of ROS such as hydrogen peroxide (H_2O_2), superoxide ($O_2^{\cdot-}$) and the hydroxyl radical ($HO^{\cdot-}$) and reactive nitrogen species such as nitric oxide ($NO^{\cdot}$) upon wounding in conjunction with the redox control of wound healing, are features common to plants and animals. The oxidative burst can directly kill pathogens by ROS-mediated degradation of their proteins, nucleic acids and deterioration of membranes, and also by inhibiting germination of pathogen spores. For example, the application of 25 μM H_2O_2 to *Peronospora tabacina*, *Cladosporium cucumerinum* and *Colletotrichum lagenarium* completely inhibits spore germination in these pathogens (Peng and Kuć, 1992). A physical barrier against invading pathogens through ROS-facilitated lignification and suberinization is created at the wound site, thereby restricting the spread of an infection. The activation of systemic wounding responses, which depend on cell-to-cell signal transduction, prevents further damage to remote tissues (Suzuki and Mittler, 2012). Systemic responses involve defence proteins, the so-called defensins, which accumulate in injured leaves and also in distant intact leaves (Green and Ryan, 1972). According to León et al. (2001), in plants, wound-inducible proteins may perform functions such as repairing of damaged tissue, producing substances to prevent pathogen infection, participating in the wound defence signalling pathways, or adjusting plant metabolism to the altered nutritional demands. Peroxidases seem to be involved in almost all of these functions. The control of ROS levels generated by wound-affected cells by antioxidant systems, including peroxidases, is key to the role of these oxidoreductases in the "plant immune system" (Almagro et al., 2009), contributing to the early stress response, signalling, healing or cell death processes.

This special issue of Phytochemistry is dedicated to Professor Paul Bolwell, a pioneer in the area of the stress-induced oxidative burst in plants. Paul was the first to demonstrate the contribution of cell wall peroxidases in the pathogen-induced oxidative burst (Bolwell et al., 1995; Robertson et al., 1995; Bolwell, 1996). We now know that an increase in the activity of apoplastic peroxidases is an early response of plants to biotic and abiotic stresses (Penel and Dunand, 2009; Daudi et al., 2012; O'Brien et al., 2012a). During the oxidative burst apoplastic peroxidases can contribute to the formation of ROS in the presence of suitable reductants (Bolwell et al., 1999, 2002). Here, we review the current knowledge of the roles of peroxidases in wounding responses. We will consider responses to wounding in model systems, such as excised roots or leaves, isolated seed axes, and sections of stems and tubers. The mechanisms of the regulation of peroxidase activity, the involvement of peroxidases in ROS production and detoxification following wounding, possible sources of peroxidases released into the apoplast and their interplay with other apoplastic redox enzymes, and the potential contribution of peroxidase-mediated redox reactions in wound healing and cell death in plants will be discussed.

2. Wounding-induced peroxidases in model systems

Among the apoplastic enzymes involved in the plant immune system, class III peroxidases (EC 1.11.1.7) play crucial roles. Class III peroxidases represent large multigene families in all land plants. Generally they are glycoproteins, contain protohaemin IX (haem b) as the prosthetic group, and are secreted into the cell wall, the apoplast or the vacuole (Mathé et al., 2010). They comprise around 350 amino acids, all have a signal peptide, and some conserved amino acid motifs, especially in the region of interaction with the heme (Passardi et al., 2004). Class III peroxidases contain eight conserved cysteines and one or several glycosylation sites. *Arabidopsis thaliana* has 73 genes that encode a peroxidase (Tognolli et al., 2002) and *Oryza sativa* has 138 genes (Passardi et al., 2004). These enzymes show a broad range in their substrate requirements and catalyze the reduction of H_2O_2 coupled with the oxidation of various electron donors, e.g. phenolic compounds, alkaloids, or auxins. The large number of different peroxidase isoforms may each have different catalytic properties owing to differences in the amino acid sequences interacting with the prosthetic group and the substrate access channel (Veitch, 2004). With broad substrate specificities, diverse protein structure and variable catalytic properties, these heme-containing enzymes have a versatile nature, and as a result, they are involved in both H_2O_2 detoxification in their regular peroxidative cycle and also the formation of ROS within a separate oxidative cycle (Mathé et al., 2010). Table 1 presents examples of recent publications implicating peroxidases in the wounding response; earlier literature is summarized in Yoshida et al. (2003). In the field, wounding may occur in various ways, but in the laboratory is often simulated by cutting plant tissues with a blade. Exactly how plants perceive wounding stress is poorly understood. Interestingly, even non-wounding mechanical stress by gentle touch can induce redox activity (Benikhlef et al., 2013). Wounding one leaf can result in an increase in peroxidase activity in adjacent, non-wounded, leaves (e.g. An et al., 2009). An early response to wounding is the release of peroxidases into the apoplast (Minibayeva et al., 2009), which is also well documented for plant responses to pathogens (e.g. Lehtonen et al., 2009). Importantly, for a particular apoplastic enzyme to be present at the correct site at the right time to perform its function depends not only on gene transcription and protein synthesis, but also secretion and targeting to the site of action (Penel and Dunand, 2009). Upon wounding, the immediate increase in peroxidase activity usually continues for at least several hours, and often precedes the accumulation of peroxidase transcripts (see Section 3) (Holm et al., 2003). This suggests that the release of peroxidases into the apoplast and their activation by post-translational modifications is responsible for the immediate increase in wound-induced activity.

It has been suggested that the release of apoplastic peroxidases is mediated by changing environmental conditions (Fecht-Christoffers et al., 2003). Apparently the cell wall has an enormous capacity to accumulate various peroxidase isoforms. In cultured *Catharanthus roseus* cells, only 4% of the total peroxidase activity was secreted in the growth medium compared to 45% by protoplasts (Mera et al., 2003). The spatial control of peroxidase secretion has been observed, for example, at the site of microbe invasion (Bestwick et al., 1997), but the mechanism has not yet been elucidated. Some apoplastic peroxidases exhibit a strong ability for the Ca^{2+}-mediated conformation of pectin (Carpin et al., 2001). This could provide a mechanism to attract peroxidases to particular sites of the cell wall, where they could produce or detoxify ROS (Delannoy et al., 2003). Calcium-binding motifs are highly conserved in class III peroxidases (Cosio and Dunand, 2009). Exogenous Ca^{2+} application can stimulate the activity of purified apoplastic peroxidases (Minibayeva et al., 2009; Plieth and Vollbehr, 2012). Plasma membrane-bound peroxidases of maize roots have two putative calcium-binding sites (Mika et al., 2008). Furthermore, it was shown that an extracellular peroxidase isolated from *Euphorbia latex* has an affinity for calmodulin, a Ca^{2+}-dependent regulatory protein, and is strongly activated by the simultaneous presence of calmodulin and Ca^{2+} (Mura et al., 2005). Secretion of peroxidases is unlikely to result from passive release from the wound surface or through damaged plasma membranes. After the excision of roots from wheat seedlings, leachates contained only

Please cite this article in press as: Minibayeva, F., et al. Roles of apoplastic peroxidases in plant response to wounding. Phytochemistry (2014), http://dx.doi.org/10.1016/j.phytochem.2014.06.008