

# Callose

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Callose is a  $\beta$ -(1,3)-D-glucan polysaccharide with some  $\beta$ -1,6-branches that exists in all multicellular green algae and higher plants.

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## 1. Introduction

Callose is a  $\beta$ -(1,3)-D-glucan polysaccharide with some  $\beta$ -1,6-branches that exists in all multicellular green algae and higher plants <sup>[1]</sup>. In most plants, callose is synthesized by a family of callose synthases and plays important roles in several important biological processes in the plant. During cell division, callose is transiently deposited in the cell plate of the cells undergoing cytokinesis <sup>[2][3]</sup>. Callose is also a critical component of the transient cell wall surrounding pollen mother cells, the four microspores after meiosis and the pre-cell wall of the growing pollen tube tip <sup>[1][4]</sup>. Callose is also present in sieve plates, a basic component of the phloem, under normal growing and developmental conditions and can accumulate rapidly and plugs the sieve pores when subjected to stress <sup>[5][6]</sup>. Similar to this stress response, callose biosynthesis and degradation in the neck region of plasmodesmata help to regulate permeability during abiotic and biotic stresses <sup>[7]</sup>. In addition, callose is deposited between the plasma membrane and the pre-existing cell wall at sites of pathogen attack <sup>[8]</sup>. This pathogen-induced callose deposition functions as a chemical and physical defense mechanism for reinforcing plant cell wall and plays an essential role in the defense response to invading pathogens. Over the past 20 years or so, a large number of studies have been published on defense-related callose deposition that address the dynamic nature of pathogen-induced callose deposition, its important roles in plant defense responses, and the complex regulatory mechanisms of defense-related callose deposition.

## 2. Defense-Related Callose Deposition in Plants

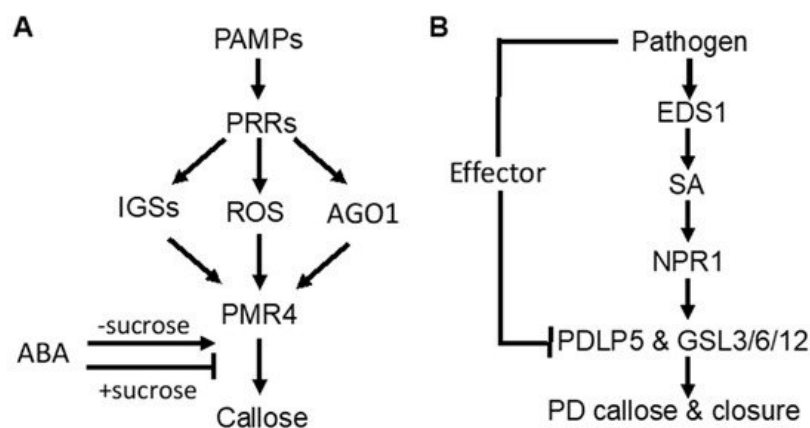
Plants are constantly exposed to a large number of microbial pathogens and have evolved complex disease resistance mechanisms that protect plants from pathogens. The first common mechanism of plant disease resistance is through pre-formed structures and compounds such as plant cuticle surfaces, plant cell walls, antimicrobial chemicals and peptides, and other arsenals that inhibit or block pathogen-derived toxins, enzymes, or other activities <sup>[9]</sup>. The second common disease resistance mechanism is an infection-induced response of the interconnected two-tier immunity system <sup>[10]</sup>. The first layer is triggered upon recognition of pathogen-associated molecular patterns (PAMPs) by plant plasma membrane-localized pattern recognition receptors (PRRs). Well-characterized PRRs include *Arabidopsis* FLS2 (Flagellin-sensing 2) and EFR (the EF-Tu receptor), which recognize bacterial flagellin and the elongation factor-Tu (EF-Tu), respectively <sup>[11]</sup>. Adapted pathogens deliver effector proteins to plant cells to suppress PAMP-triggered immunity (PTI) <sup>[12]</sup>. To counter this, plants have evolved the second layer of the immunity system through recognition of pathogen effectors to activate effector-triggered immunity (ETI). Receptors for ETI are often plant resistance (R) proteins containing nucleotide-binding site (NBS) and leucine-rich repeat (LRR) domains with a Toll-interleukin receptor (TIR) or coiled-coil (CC) N-terminal domain <sup>[13]</sup>. Both PTI and ETI are associated with diverse signaling processes including dynamic protein interactions and phosphorylation, generation of reactive oxygen species (ROS),  $\text{Ca}^{++}$  signal spike, and mitogen-activated protein kinase (MAPK) activation <sup>[14]</sup>. These signaling processes then converge in the nucleus to elicit host transcriptional reprogramming not only for defense but also for balancing defense with growth <sup>[15]</sup>.

In *Arabidopsis*, there are 12 genes encoding GLUCAN SYNTHASE-LIKE (GSL) callose synthases, and pathogen- and PAMP-induced callose deposition is dependent on the GSL5/(POWDERY MILDEW RESISTANT 4 (PMR4) callose synthase <sup>[16]</sup>. Callose-induced PAMPs include the 22-amino acid sequence of the conserved N-terminal part of flagellin (Flg22) and the 18 amino acid peptide of the bacterial elongation factor EF-Tu (Elf18) <sup>[17]</sup>. Fungal cell elicitors including chitin, a  $\beta$ -(1,4)-linked polymer of *N*-acetylglucosamine, and chitosan, a randomly distributed  $\beta$ -(1,4)-linked polymer of D-glucosamine and acetylglucosamine, are also potent PAMPs for inducing callose deposition <sup>[18]</sup>. Besides PAMPs,

endogenous damage-associated patterns (DAMP) such as oligogalacturonides from pathogen- or herbivore-damaged plant tissues can function as elicitors to activate callose depositions as well [19]. Pathogen-induced callose can also be primed or enhanced by prior pathogen infection or defense-inducing compounds. For example, induction of systemic acquired resistance (SAR) by local pathogen infection is associated with augmented levels of callose upon secondary pathogen inoculation [20]. Furthermore, resistance-inducing chemicals including salicylic acid (SA), SA analog benzo(1,2,3) thiadiazole-7-carbothioic acid S-methyl ester (BTH), and the nonprotein amino acid BABA can augment depositions of pathogen-inducible callose [21][22]. It has also been recently reported that in tomato and wheat callose priming can be induced by *Rhizophagus irregularis*, a mycorrhizal fungus that can enhance resistance to several pathogens such as the necrotrophic fungal pathogen *Botrytis cinerea* in tomato [23][24].

One of the earliest plant defense responses against haustorium-forming pathogens is the deposition of a cell wall-associated apposition called papillae for halting the fungal penetration attempt [8]. If the fungal pathogen is successful in penetration and forms a haustorial feeding structure, the cell wall apposition materials can form collars or neck bands, including partial or even full encasements around the haustorium [25]. Common biochemical constituents of papillae include callose, phenolic, and phenolic conjugates such as phenolic-polyamines, ROS, peroxidases, cell wall structural proteins such as arabinogalactan proteins and hydroxyproline-rich glycoproteins, and cell wall polymers including pectin and xyloglucans [25]. Immuno-fluorescence and immune-gold labeling also found that *Arabidopsis* encasements surrounding the haustoria of the powdery mildew fungal pathogen *Golovinomyces orontii* contain callose, arabinogalactan proteins, rhamnogalacturonanI, a  $\beta$ -linked galactose-containing protein, and xyloglucanin [26], supporting the idea that the papillae and encasements are related and their formation is associated with increased callose deposition.

Callose deposition is also induced as a defense mechanism in cell wall near the neck zone of plasmodesmata to control their permeability [27]. Plasmodesmata are symplastic junctions between cells that function as important pathways for intercellular communication and molecular exchange, including cell-to-cell spread of viruses [28][29]. The level of callose in the plasmodesmatal neck zone is important for the cell-to-cell movement of cellular molecules and viruses; high levels of callose reduce or even close plasmodesmatal channels, while low levels of callose open them [28][29] (Figure 1). The level of callose in the plasmodesmata is regulated by two groups of enzymes; callose synthases synthesize callose while  $\beta$ -1,3-glucanases degrade it [28][29]. Callose accumulation in the plasmodesmata is induced by many viruses including *Tobacco mosaic virus* (TMV), *Maize dwarf mosaic virus* (MDMV), *Potato virus X* (PVX), and *Tomato bushy stunt virus* (TBSV) [7][28][29][30].



**Figure 1.** Signaling of pathogen-induced callose deposition in plants. **(A)** pathogen-associated molecular pattern (PAMP)-induced callose deposition is mediated by pattern-recognition receptors (PRRs) and promoted by indole glucosinolates (IGSs), reactive oxygen species (ROS), and RNAi regulatory protein Argonaute1 (AGO1). ABA stimulates but represses PAMP-induced callose deposition in the presence of high sucrose concentrations. **(B)** pathogen-induced callose deposition in plasmodesmata (PD) through the SA-mediated signaling pathway.

## 3. Regulation of Defense-Related Callose Deposition

### 3.1. Signaling Pathways Controlling Pathogen-Induced Callose Deposition

As part of PTI, PAMP-induced callose deposition is under the control of plant PRRs. Activity of the downstream pathways is marked by common signaling events, such as anion fluxes, protein phosphorylation cascades, accumulation of ROS, and defense gene induction. In *Arabidopsis*, ROS act as positive signals in Flg22-induced callose [31][32] (Figure 1A). The RNAi regulatory protein Argonaute1 also generates various miRNA signals that stimulate or repress Flg22-induced callose [33] (Figure 1A). Flg22-induced callose in *Arabidopsis* also requires intact biosynthesis of 4-methoxylated indole

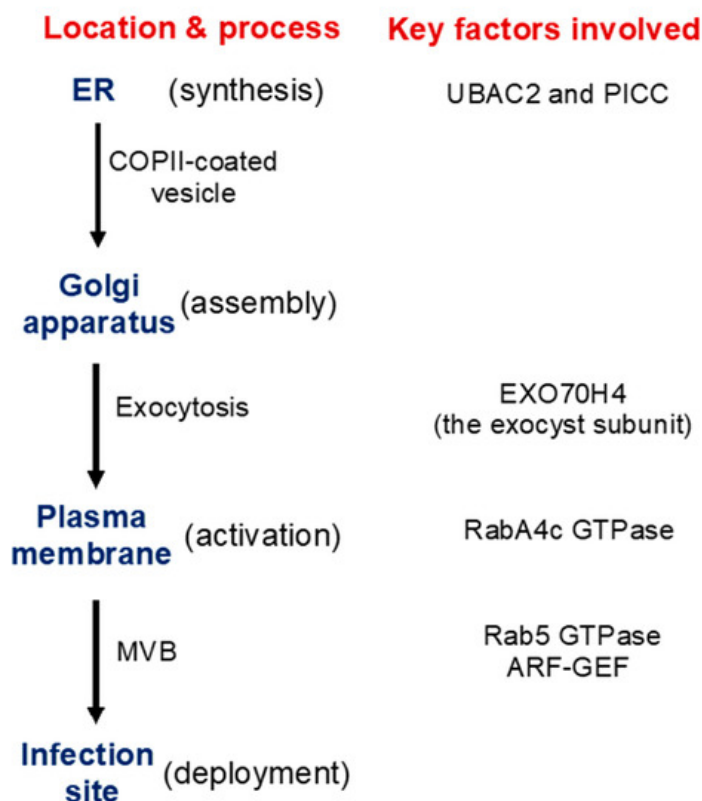
glucosinolates<sup>[34]</sup> (Figure 1A), suggesting that these secondary metabolites or break-down products play a crucial role in the regulation of callose. Interestingly, Flg22- and chitosan-induced callose differ in the requirement for the NADPH oxidase RBOHD, the glucosinolate regulatory enzymes PEN2, and the callose synthase PMR4<sup>[35]</sup>. The *rbohD* mutant accumulated reduced levels of Flg22-induced H<sub>2</sub>O<sub>2</sub> and failed to deposit enhanced levels of callose upon treatment with Flg22. However, the *rbohD* mutant deposited normal levels of callose in response to chitosan, even though chitosan-induced H<sub>2</sub>O<sub>2</sub> was reduced. Likewise, the *pen2-2* mutant failed to deposit enhanced callose in response to Flg22, whereas chitosan induced statistically significant enhancements in callose deposition. The *pmr4-1* mutant deposited dramatically reduced levels of basal callose and failed to respond to Flg22. However, chitosan still elicited a residual callose response in *pmr4-1* plants, indicating that one or more other callose synthases than PMR4 are responsible for a portion of chitosan-induced callose. These results demonstrated that PAMP-induced callose is controlled by distinct signaling pathways.

SA plays an important role in pathogen-induced plasmodesmata closure and associated callose deposition in *Arabidopsis*<sup>[36]</sup>. Direct exogenous application of SA or bacterial infection induce plasmodesmata closure and callose deposition. SA pathway mutants are impaired in this response. The SA- or pathogen-induced plasmodesmata closure and associated callose deposition requires an ENHANCED DISEASE RESISTANCE1(EDS1)- and NONEXPRESSOR OF PATHOGENESIS-RELATED GENES1 (NPR1)-dependent SA pathway and is dependent on the regulator of plasmodesmal gating PLASMODESMATA-LOCATED PROTEIN5(PDL5)<sup>[36]</sup>(Figure 1B). These results indicate that SA is a crucial signal in the regulation of cell-to-cell communication and trafficking via plasmodesmata in plant cells during innate immune responses.

Abscissic acid (ABA) signaling has an important effect on induced or primed callose deposition. Callose priming induced by the nonprotein amino acid  $\beta$ -amino butyric acid (BABA) requires an intact ABA-dependent pathway in *Arabidopsis*. BABA-induced resistance in *Arabidopsis* against *Plectosphaerella cucumerina* is known to be mediated by callose priming. Indole-3-carboxylic acid (ICOOH, also known as I3CA) mediates BABA-induced resistance in *Arabidopsis* against *P. cucumerina*. I3CA treatment increased ABA levels, which activates a starch amylase (BAM1) to trigger augmented callose deposition against *P. cucumerina*<sup>[37]</sup>. A similar role of ABA and starch metabolism has also been reported in mycorrhiza-induced callose priming in tomato plants upon infection by necrotrophic fungal pathogen *B. cinerea*<sup>[24]</sup>. Intriguingly, ABA can enhance or repress Flg22-induced callose deposition in *Arabidopsis* depending on growth conditions<sup>[35]</sup> (Figure 1A). Under most growth conditions, ABA has a stimulatory effect on Flg22-induced callose deposition. However, ABA reduced basal and PAMP-induced callose at low light intensity, high sucrose concentration and vitamins (Figure 1A). ABA also represses Flg22-induced callose in hydroponic *Arabidopsis* grown with the a relatively high concentration of sucrose in the growth medium (0.5%)<sup>[34][35]</sup> (Figure 1A). These results suggest that ABA's role in defense-related callose deposition and priming is influenced by carbohydrate metabolism.

### 3.2. Biogenesis and Activation of Pathogen-Responsive Callose Synthases

As a large transmembrane protein, GSL5/PMR4 belongs to the family of glucan synthases that include plant cellulose synthases. Plant cellulose synthases are synthesized on the endoplasmic reticulum (ER) and go through the ER quality control system for proper folding and modification before being assembled into complexes in the Golgi apparatus and transported to the plasma membrane through the Golgi- and post-Golgi trafficking system<sup>[38]</sup>. Many proteins involved in plant immune responses, including pattern-recognition receptors and extracellular defense-related proteins, are also synthesized on the ER, go through the ER quality control, and ultimately are transported to their destinations through the secretory pathway. We have recently shown that two *Arabidopsis* homologs of UBAC2, a conserved ER protein implicated in ER quality control and ER-associated degradation (ERAD), play a critical role in pathogen- and PAMP-induced callose deposition<sup>[39]</sup>. The UBAC2 proteins interact with the plant-specific PAMP-INDUCED COILED COIL (PICC) protein, which is also localized in the ER and is important for pathogen- and PAMP-induced callose deposition (Figure 2). Therefore, the evolutionarily conserved UBAC2 and plant-specific PICC proteins are critical components of an ER pathway with an important role in plant immunity by regulating pathogen-induced callose deposition. The compromised phenotypes of the *ubac2* and *picc* mutants in PTI and pathogen/PAMP-induced callose deposition could be rescued by overexpression of PMR4, suggesting that disruption of UBAC2 and PICC genes may reduce the accumulation of PMR4 callose synthase in the *ubac2* and *picc* mutants, leading to defects in pathogen/PAMP-induced callose deposition<sup>[39]</sup>. Further analysis using both GFP- and myc-labeled PMR4 indeed discovered that disruption of UBAC2 or PICC reduced the levels of plasma-membrane-localized PMR4<sup>[39]</sup>. These results indicate that the evolutionarily conserved UBAC2 proteins function in coordination with a plant-specific protein PICC in the positive regulation of the biogenesis of PMR4, either by stabilizing the callose synthase or degrading a negative regulator of PMR4 stability (Figure 2).



**Figure 2.** Synthesis, transport, activation, and recruitment of *Arabidopsis* PMR4 callose synthase during plant defense responses. Some of the important factors identified to be involved in these processes are indicated.

In addition, there is evidence for PMR4 activation in response to pathogen infection or other stress signals. In uninfected *PMR4*-overexpression lines, no significant increase in the callose synthase activity or callose deposition was observed [40]. In yeast, activation and translocation of callose synthases involve GTPases [41][42]. In *Arabidopsis*, PMR4 interacts with GTPase RabA4c [43] (Figure 2). Similar to PMR4 overexpression, RabA4c overexpression leads to complete penetration resistance to the virulent powdery mildew fungal pathogen in a PMR4-dependent manner [43]. By contrast, overexpression of a dominant negative form of RabA4c fails to increase callose deposition or penetration resistance [43]. These results indicate that the RabA4c GTPase plays a critical role in the activation and translocation of PMR4 during pathogen-induced callose deposition. As a transmembrane protein synthesized on the ER, PMR4 is likely subjected to additional regulatory mechanisms for its biogenesis, trafficking, and activation to ensure biosynthesis of callose in a timely manner.

### 3.3. Transport Processes in Pathogen-Induced Callose Deposition

Exocytosis is a process by which cells are able to not only move materials from within a cell to the exterior of the cell, but also insert membrane proteins (such as ion channels and cell surface receptors), lipids, and other components into the cell membrane [44]. Vesicles containing these membrane components fully fuse with and become part of the plasma membrane. The exocyst, an octameric protein complex involved in exocytosis, tethers and spatially target post-Golgi vesicles to the plasma membrane prior to vesicle fusion [44]. It has been demonstrated that the exocyst subunit EXO70H4 is important in the deposition of the callose-rich cell wall layer for biogenesis of the trichome secondary cell wall [45][46]. PMR4 is a callose synthase responsible for the synthesis of callose in the trichome and PMR4 colocalizes with EXO70H4 on plasma membrane microdomains that do not develop in the *exo70h4-1* mutant. Significantly, *EXO70H4* expression is induced by pathogen elicitor Flg22, which induces callose deposition, in epidermal pavement cells [46]. These results raise a strong possibility that EXO70H4-dependent exocytosis is also involved in the trafficking of newly synthesized PMR4 callose synthase proteins from the Golgi apparatus to the plasma membrane (Figure 2).

Another transport process important for defense-related callose deposition is the recruitment of callose synthases protein such as PMR4 in *Arabidopsis* from the plasma membrane and transport in vesicle-like bodies to the site of attempted penetration after fungal infection (Figure 1). There is strong evidence that multivesicular bodies (MVBs) are involved in the transportation and delivery of defense components to the forming papilla. Studies have showed accumulation of MVBs and cell wall-associated paramural bodies (PMBs) in the vicinity of pathogen-induced papillae [47][48][49]. PMBs, which are situated between the cell wall and the plasma membrane, are likely to have resulted from the fusion of MVBs with the plasma membrane [50]. Plant MVBs and PMBs have been observed near papillae in plant cells infected by pathogenic fungal, bacteria and nematodes for delivery of defense-related molecules including phytoalexins, callose and ROS to

papillae [47][48][51][52][53]. In addition, in plant interaction with filamentous pathogens, each haustorium formed upon successful penetration is surrounded by the plasma membrane of the plant cell termed extrahaustorial membrane (EHM), which is likely synthesized de novo [54]. In tobacco (*Nicotiana benthamiana*) cells invaded by oomycete pathogen *P. infestans*, the Rab7 GTPase RabG3c MVB marker protein, but not a tonoplast-localized sucrose transporter, is recruited to the EHM [55]. In *Arabidopsis*, the Rab5 GTPase, also an MVB marker, accumulates in the EHM after infection with a powdery mildew fungus [56] (Figure 2). Thus, specific rerouting of MVBs from the vacuole to the host-pathogen interface may participate in the formation or modulation of the EHM. In barley, MVBs contained the ADP-ribosylation factor (ARF) GTPase ARFA1b/1c that was important for callose deposition at powdery mildew penetration sites [51]. RNAi knockdown or expression of a dominant negative ARFA1b/1c variant abolished callose accumulation at penetration sites and resulted in increased fungal penetration success. ARFA1b/1c localized to an endosomal MVB compartment that accumulated at fungal penetration sites prior to the accumulation of callose. Interestingly, the *Arabidopsis* ARF-GEF (guanine nucleotide exchange factor) MIN7 is required for normal levels of callose deposition in response to the *Pseudomonas syringae* pv. *tomato* ΔCEL mutant, suggesting that an ARF-dependent vesicle trafficking process may also play a role in callose deposition at sites of pathogen detection in *Arabidopsis* [57] (Figure 2).

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