Aluminum-Induced Inhibition of Root Growth: Roles of Cell Wall Assembly, Structure, and Function

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Abstract Aluminum (Al) toxicity is the most important soil constraint for plant growth and development in acid soils. It is a matter of debate whether the primary lesions of Al toxicity are apoplastic or symplastic, while there is increasing physiological, biochemical, and molecular evidence showing that the modification of cell wall properties contributes to the Al-induced inhibition of root growth. The rapid binding of Al in the root cell wall particularly to the pectin matrix and hemicellulose can affect cell wall properties. Most recent studies have revealed that the local accumulation of auxin in the most Al-sensitive root zone of the root apex is a major factor leading to Al-induced root-growth inhibition. Evidence suggests that the auxin effect is mediated mainly via modification of cell wall structural properties. A further in-depth characterization of the Al-induced apoplastic reactions in the most Al-sensitive zone of the root apex is urgently required to better understand the phytohormone-mediated signaling network leading to Al-induced inhibition of root growth.

1 Introduction

Soil acidity with $pH \leq 5.5$ is one of the most important factors limiting crop production worldwide on approximately 30 % of the world's total land area and as much as 50 % of the world's potentially arable lands. The tropics and subtropics account for 60 % of the acid soils in the world. In tropical areas, about 43 % of soils are acidic comprising about 68 % of tropical America, 38 % of tropical Asia, and 27 % of tropical Africa (von Uexküll and Mutert 1995). When the soil pH drops below 5, Al^{3+} is solubilized into the soil solution and becomes a major constraint for

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S.K. Panda, F. Baluška (eds.), *Aluminum Stress Adaptation in Plants*, Signaling and Communication in Plants 24, DOI 10.1007/978-3-319-19968-9_13

plant growth and development in acidic soils (Kinraide et al. 1992). The easily observable symptom of Al toxicity is a rapid (minutes to few hours) inhibition of root growth (Horst et al. 1992; Delhaize and Ryan 1995), resulting in a reduced and damaged root system that limits mineral nutrient and water uptake (Kochian et al. 2004). The rapidity of this response indicates that Al first inhibits root cell expansion and elongation and consequently cell division over the longer term (Delhaize and Ryan 1995; Kochian 1995). Another most sensitive indicator of Al injury on roots is the induction of callose synthesis (Wissemeier et al. 1987), particularly in the root apex (Wissemeier and Horst 1995).

Although much progress has been made during recent years in the understanding of Al resistance, the molecular and physiological mechanisms leading to Al-induced inhibition of root elongation are still not well understood. There are a number of excellent reviews in recent years summarizing the state of knowledge and addressing knowledge gaps (Horst 1995; Kochian et al. 2004; Panda and Matsumoto 2007; Panda et al. 2009; Horst et al. 2010; Delhaize et al. 2012; Liu et al. 2014; Kochian et al. 2015). Particularly, the relative importance of symplastic versus apoplastic lesions of Al toxicity remains a matter of debate. The studies by Zheng et al. (Yang et al. 2008, 2011; Zhu et al. 2012) and especially by Horst et al. (2010) focused on the attention on the role of the apoplast in Al toxicity regarding short-term inhibition of root elongation by Al. Here we summarize the current understanding of the role of root cell wall structure and assembly in Al-induced inhibition of root growth of plants.

2 Al Toxicity Is Targeted Primarily to the Root-Apex Transition Zone Whereas the Elongation Zone Is Not, or Less, Affected Depending on Plant Species

The inhibition of root growth has been established as the main symptom of Al toxicity in barley (*Hordeum vulgare*) and rye (*Secale cereale*) early in 1918 (Hartwell and Pember 1918). Seventy-five years after this finding, Ryan et al. (1993) confirmed that the root apex is the major perception site of Al toxicity in maize (*Zea mays*). The root apex of higher plants is quite sensitive to environmental stimuli. As the most prominent plant organ, the root cap can sense diverse physical and chemical stimuli such as gravity, light, humidity, oxygen, and mineral elements. Subsequently, the motoric responses to these stimuli are transmitted to the elongation zone (Baluška and Mancuso 2013). The transition zone, which is located between the apical meristem and basal elongation zone of the root, has a unique role as the determiner of cell fate and root growth (Baluška et al. 2010). In Lixis, an Al-sensitive genotype of maize, Sivaguru and Horst (1998) specified that the distal part of the transition zone (DTZ, 1–2 mm) is the most Al-sensitive zone of the root apex. Application of Al to the DTZ but not the elongation zone (EZ) reduced cell elongation in the EZ to the same extent as application to the entire 10 mm root apex (Fig. 1). The transition zone (TZ) of the root apex as the

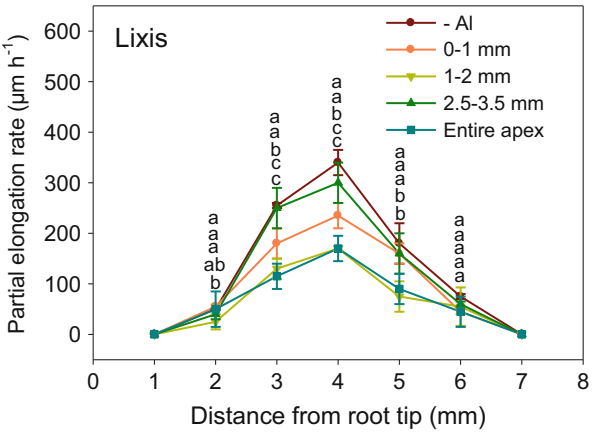

Fig. 1 Effect of Al supply (90 µM, 1 h) to the entire root apex or specific 1-mm apical root zones on partial elongation rates of apical 1-mm root segments of the primary root of the maize cv Lixis (Al-sensitive). Values are means of five independent measurements ±SD. Different letters indicate significant differences at $p < 0.05$ (Tukey test). From Kollmeier et al. (2000)

primary Al-toxic site of plants has been also proposed in other species, such as *Arabidopsis thaliana* (Illéš et al. 2006; Yang et al. 2014) and *Sorghum bicolor* (Sivaguru et al. 2013). While in common bean (*Phaseolus vulgaris*), a leguminous plant, Rangel et al. (2007) showed that in addition to the TZ also the EZ of the root apex responds to Al exposure. This difference might be due to the fact that dicotyledons and grasses (*Poales*) are different in the composition of their cell walls. Particularly, the pectin content of the cell walls is higher in dicotyledons (Carpita and Gibeaut 1993) which explains their enhanced Al-binding capacity. The important role of the cell wall pectin content for Al accumulation and Al sensitivity has been demonstrated repeatedly (Horst et al. 2010).

Cells in the TZ are very active in cytoskeletal rearrangements, in endocytosis and endocytic vesicle recycling, as well as in electric activities (Baluška and Mancuso 2013). In Arabidopsis, Illéš et al. (2006) found that Al can rapidly depolarize the plasma membrane, while the full recovery of the membrane potential was slower in the cells of the DTZ than in the proximal transition zone (PTZ) after the removal of external Al stress, implying that the DTZ is the most Al-sensitive site in the root apex. Further combination of morin staining to detect Al accumulation in cells and FM4-64 for endosomal/vacuolar membrane observation was used; these authors found that the Al internalization mainly occurred in the cells of the DTZ rather than the proximal transition zone and EZ. Using monoclonal tubulin and actin antibodies, Sivaguru et al. (1999) found that the more sensitive response to Al of root elongation in the DTZ results from a higher Al accumulation in this zone accompanied by Al-mediated alterations to microtubules and actin microfilaments, Al-induced depolarization of the plasma membrane, and callose formation particularly in the outer cortex cells of the DTZ (Sivaguru and Horst 1998). It is possible that the rapid Al-induced changes to cytosolic Ca^{2+} ($[Ca^{2+}]_{cyt}$) may mediate cytoskeletal disorders, since Rincón-Zachary et al. (2010) using fluorescence resonance energy transfer (FRET)-sensitized emission to image *Arabidopsis thaliana* roots expressing the yellow cameleon 3.60 Ca^{2+} reporter demonstrated that Al evoked an increase of $[Ca^{2+}]_{cyt}$ within seconds primarily in

the TZ of the root apex. The elevated $[Ca^{2+}]_{cyt}$ and modification of the plasma membrane are known to be crucial for callose deposition (Kauss et al. 1990) and the effect of Al on actin microfilaments via involvement of Ca^{2+}-mediated kinases and phosphatases (Grabski et al. 1998).

Exclusion of Al from the apoplast through exudation of organic acid such as citrate and malate has been identified as one of the major Al resistance mechanisms in plants (Ma et al. 2001; Kochian et al. 2004; Ryan et al. 2011). Al-activated citrate transporters (MATE) and Al-activated malate transporters (ALMT) have been identified to be crucial for citrate and malate exudation from root tips conferring Al exclusion and Al resistance (Delhaize et al. 2012). Using the laser-capture microdissection (LCM) technique, Sivaguru et al. (2013) found that Al-induced *SbMATE* gene expression and protein synthesis were specifically localized to the epidermal and outer cortical cell layers of the root-apex DTZ in the Al-resistant near-isogenic sorghum line. In this root zone, Al induced the greatest cell damage and generation of reactive oxygen species (ROS). The specific Al-induced ROS induction in the DTZ may play a signaling role in the induction of *SbMATE* gene expression. The H_2O_2-induced *ALMT1* gene expression and malate exudation from the root apex in Arabidopsis (Kobayashi et al. 2013) may support this hypothesis.

3 Auxin Is Involved in Al-Induced Inhibition of Root Elongation

3.1 Auxin Accumulation in the Transition Zone and Inhibition of Auxin Transport into the EZ Are Involved in Inhibition of Cell Elongation in the EZ

In the root-apex TZ, the meristematic cells exit the cell division phase and prepare for filamentous actin (F-actin)-dependent rapid cell elongation (Baluška et al. 1992, 2001; Verbelen et al. 2006). Cell division in the MZ and cell elongation in the EZ are inhibited as primary effects of Al occurring in the adjacent transition zone, in which these processes are less active. This observation suggests that putative primary Al signals are transduced from the transition zone to both the root meristem and the fast elongation zone. Auxin is an important regulator of root cell division, elongation, and differentiation. Hence, auxin controls overall root growth. High concentrations of auxin, however, inhibit the elongation of certain cell types (Teale et al. 2005). Auxin signaling within the root-apex TZ is sensitive not only to developmental signals but also to environmental cues (Baluška et al. 2010), including Al (Sivaguru and Horst 1998). Several studies have demonstrated that aluminum may interact with auxin signaling pathways, leading to alterations of auxin accumulation and distribution in roots (Kollmeier et al. 2000; Doncheva et al. 2005; Shen et al. 2008). In maize, local application of Al to the DTZ promoted auxin accumulation in the MZ and DTZ, while reduced auxin level in the EZ. External

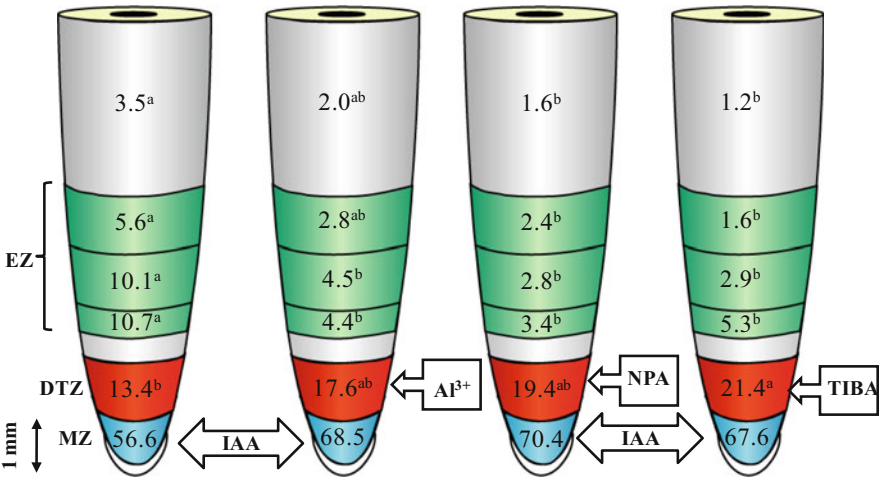

Fig. 2 Effect of application of Al and IAA transport inhibitors to the DTZ of primary roots of the maize cv Lixis (Al-sensitive) on the relative distribution of [^{3}H]IAA applied to the MZ (total uptake = 100 %). Application of 90 mM mononuclear Al, 10 mM NPA, or 10 mM TIBA in nutrient solution, pH 4.3, in 0.6 % (w/v) agarose gel to the DTZ for 30 min. Control roots were treated only with nutrient solution in agarose blocks, pH 4.3. [^{3}H]IAA (0.1 mM in 1.2 % [w/v] agarose blocks containing nutrient solution) was applied to the MZ for 30 min. Values are means of five independent replicates ±SD. Different letters indicate significant differences at $p < 0.05$ (Tukey test). From Kollmeier et al. (2000)

supply of auxin to the EZ was able to partially overcome the inhibition of root growth imposed by the application of Al to the DTZ (Kollmeier et al. 2000). The coincidence of the response to Al of auxin distribution in the root apex with the local supply of the auxin polar transport inhibitor N-1-naphthylphthalamic acid (NPA) and 2,3,5-triiodobenzoic acid (TIBA) (Fig. 2) suggested that the blockage of auxin polar transport and thus auxin signaling from the DTZ to the EZ could be the primary cause of Al-induced inhibition of root growth. Also, Doncheva et al. (2005) found that after the local application of the auxin polar transport inhibitor NPA to the DTZ of the root apex, abundant S-phase nuclei were observed in the distal elongation zone (DEZ) at 2.5–3 mm from the root tip, suggesting that the inhibition of auxin transport plays a role in Al-induced alteration of root cell patterning.

The cells of the DTZ are unique from all other root cells from the perspective of endocytosis, vesicle recycling, polar auxin transport, and as the first region of the root apex which is not covered with mucilage (Baluška et al. 2010). A relationship between Al toxicity, endocytosis, endosome, and vesicle recycling in the TZ cells of Arabidopsis roots has been demonstrated by Illéš et al. (2006). In addition, in Arabidopsis, Shen et al. (2008) have found that Al inhibited root to shoot auxin transport and thus root growth mainly through the blockage of the transport of PIN2 vesicles from plasma membrane to endosomes. However, the recent study by Yang

et al. (2014) suggests that the auxin transporter PIN1 rather than PIN2 is involved in the modulation of Al-induced inhibition of root growth.

3.2 *TAA1-Regulated Local Auxin Biosynthesis in the Root-Apex Transition Zone Mediates the Aluminum-Induced Inhibition of Root Growth in Arabidopsis*

Generally, auxin is transported to roots via a polar transport system from auxin-synthesizing shoot tissues (Petrášek and Friml 2009). However, in addition to being synthesized in the shoots, auxin is also generated in the roots (Overvoorde et al. 2010). In fact, the gradients of auxin in root apices depend on both local biosynthesis and directional intercellular auxin transport (Petersson et al. 2009; Petrášek and Friml 2009). It is becoming clear that the root-generated auxin contributes to the maintenance of the gradients and maxima required for normal root development, and many auxin biosynthesis genes have been identified in the root apex (Ljung et al. 2005; Petersson et al. 2009; Overvoorde et al. 2010). High-resolution auxin-measurement and gene-expression analysis in specific cell types after fluorescence-activated cell sorting revealed that the root apex is the site of auxin biosynthesis, and a substantial contribution of local biosynthesis to auxin homeostasis in the root tip was proposed (Petersson et al. 2009). Only recently the complete auxin biosynthesis pathway has been established: a two-step pathway concerts tryptophan (Trp) to indol-3-acetic acid (IAA) in plants, in which Trp is first converted to indole-3-pyruvate (IPA) by the TAA family of amino acid transferases and subsequently IAA is produced from IPA by the YUC family of flavin monooxygenases (Zhao 2012). The transcriptional analysis of maize roots growing in acid soil provided indirect evidence of enhanced auxin biosynthesis in Al-induced inhibition of root growth (Mattiello et al. 2010). Genes encoding enzymes involved in auxin biosynthesis such as *IAA amidohydrolase* (Zm.3056.1.A1_at) and *anthranilate phosphoribosyltransferase* (Zm.1556.1.A1_at) were upregulated in the root apex of the soil acidity-sensitive line S1587-17, while the auxin-degrading enzyme *indole-3-acetate beta-glucosyltransferase* (Zm.18805.1.A1_at) was downregulated after 3 days exposure to soil acidity. Recently, we clearly showed that the *TAA1*-regulated local auxin biosynthesis in the root-apex TZ mediates Al-induced inhibition of root growth (Yang et al. 2014), and this local induction of auxin biosynthesis depended on the ethylene signaling pathway (Fig. 3). However, we cannot exclude that other genes belonging to the YUC family could also contribute to Al-induced local auxin biosynthesis, since the mutant line of *YUC1D* also showed reduced Al toxicity (Yang et al. 2014).

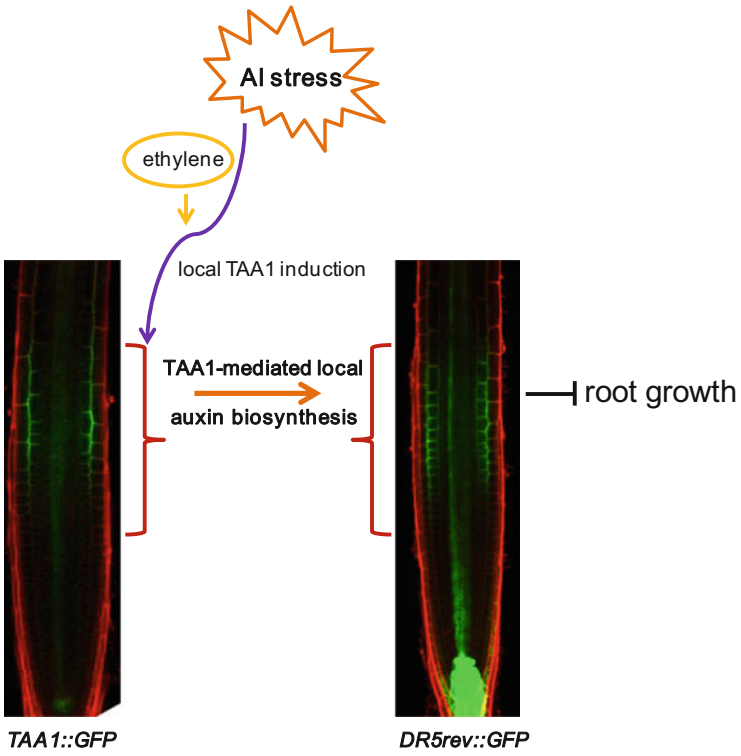

Fig. 3 Schematic representation of *TAA1*-regulated local auxin biosynthesis in the transition zone (TZ) mediating root-growth inhibition in response to Al stress. Exposure of Arabidopsis roots to Al induces a localized enhancement of auxin signaling in the root-apex TZ that is dependent on *TAA1*, which encodes a Trp aminotransferase and regulates auxin biosynthesis. *TAA1* is specifically upregulated in the root apex TZ in response to Al stress, thus mediating local auxin biosynthesis and inhibition of root growth. The *TAA1*-regulated local auxin biosynthesis in the root apex TZ in response to Al stress is dependent on ethylene. From Yang et al. (2014)

3.3 Cell Wall Modification Is a Downstream Response to Al Stress and Contributes to the Auxin-Mediated Root-Growth Inhibition

Cell walls are dynamic structures and the primary walls of plants consist of a cellulose–hemicellulose interlinked network embedded in a matrix of pectins and cell wall structure proteins (Carpita and Gibeaut 1993; Cosgrove 1997, 2005). The rapid enlargement of cells requires wall loosening, which involves modification of the molecular interactions within the CW network, resulting in the relaxation of wall tension (Perrot-Rechenmann 2010). Auxin was shown to induce rapid cell elongation in stem, coleoptile, or hypocotyl segments within minutes after auxin treatment (Rayle and Cleland 1992; Cleland 1995). According to the acid-growth

theory, this rapid effect is believed to result from the activation of a plasma-membrane H^+-ATPase, inducing extrusion of H^+ for apoplastic acidification, activation of expansins, and subsequent wall loosening (Hager 2003). Takahashi et al. (2012) showed that auxin activates the plasma-membrane H^+-ATPase by phosphorylation and regulates hypocotyl elongation in Arabidopsis. However, it appears that Al-induced inhibition of root growth can hardly be explained by reduced auxin activation of the plasma-membrane H^+-ATPase, since several studies have shown that the Al-induced plasma-membrane H^+-ATPase activity rather contributed to Al resistance (Shen et al. 2004; Yang et al. 2007; Chen et al. 2013). However, the possibility that auxin-induced excess acidification in the cell wall leading to the inhibition of root growth under Al stress cannot be ruled out.

Compared to the hypothesis of cell wall acidification, it is more probable that auxin mediates Al-induced inhibition of root growth directly by interaction with CW proteins through auxin-responsive factors (ARFs) (Fig. 4). The transcriptomic analysis presented by Yang et al. (2014) revealed that many of the differentially transcribed genes associated with cell wall modification were regulated by the transcription factors ARF10 and ARF16, suggesting that the auxin-regulated Al-induced inhibition of root growth arises from auxin signaling regulated modification of cell wall structure and/or structural components. Pitaksaringkarn et al. (2014) found that auxin regulates *XTH19* and *XTH20* expression, which are involved in cell proliferation in incised Arabidopsis inflorescence stems. However, the study by Zhu et al. (2013) revealed that auxin enhances Al toxicity via an alteration of *ALUMINUM-SENSITIVE1*-mediated Al distribution in the symplast. In spite of this, the recent study by Wu et al. (2014) in rice indicates that overexpression of *OsPIN2* alleviates the Al-induced cell rigidity in the root apex by modulating PIN2-based auxin transport, IAA efflux, and CW acidification. The reduction of Al accumulation mainly in the CW of the *OsPIN2* overexpression line further supports the hypothesis that the CW modification is probably a downstream response to Al exposure and contributes to the auxin-mediated root-growth inhibition by Al stress.

3.4 *Al Toxicity Requires Al Accumulation/Binding in the Cell Wall*

The accumulation of Al in root tips is characterized by a rapid initial phase and a low rate at later stages (Zhang and Taylor 1989, 1990). The rapid initial phase reflects the binding of Al in the apoplast (Taylor et al. 2000; Wang et al. 2004; Horst et al. 2007; Rangel et al. 2009) in which the negatively charged carboxylic groups of pectin provide the Al^{3+}-binding sites (Blamey et al. 1990; Chang et al. 1999). In fact, the involvement of pectin in Al resistance mainly depends on its degree of methylation (DM), since the DM is responsible for the negativity of the CW (Eticha et al. 2005), which is controlled by pectin methylesterase (PME) (Bordenave 1996;

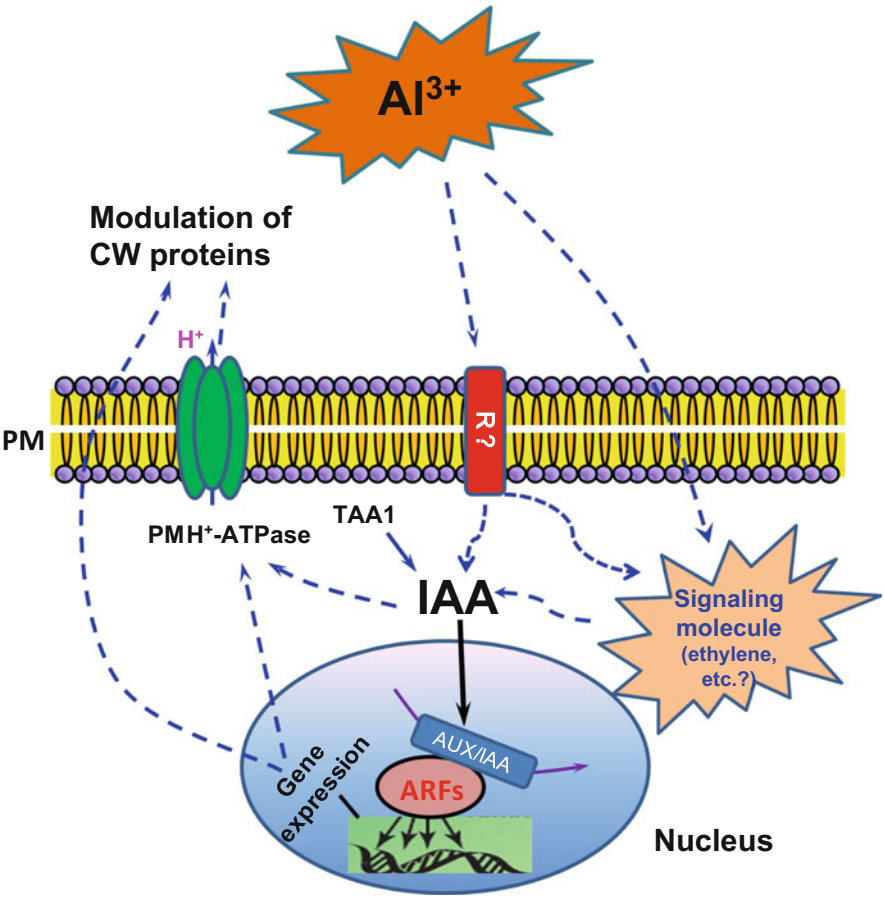

Fig. 4 Hypothetical scheme of the regulatory role of IAA in Al-induced inhibition of root growth via modification of cell wall (CW) properties. Al rapidly triggers a signaling pathway through acting on an unknown receptor (R) localized at the plasma membrane (PM), or through a signaling molecule such as ethylene, etc. Consequently, the downstream signaling of TAA1-regulated synthesized auxin is activated, in which auxin-responsive factors (ARFs) directly or indirectly regulate CW proteins or activate gene expression or activity of the PM H^+-ATPase modulating the activities of CW proteins through changes of the apoplastic pH

Gerendás 2007). In potato (*Solanum tuberosum* L.), higher Al accumulation and callose production in the roots and more severe inhibition of root growth were found in transgenic plants with higher PME expression than the wild type when exposed to Al (Schmohl et al. 2000). Short-term PME treatment of intact maize roots enhanced Al accumulation and Al-induced inhibition of root elongation (Horst et al. 2007). In two differential Al-resistant cultivars of maize, Eticha et al. (2005) observed that the Al-sensitive cultivar had lower DM and greater Al accumulation, and thus were more severely injured by Al compared with the Al-resistant cultivar, while no difference was found in pectin content. Similarly,

in rice (*Oryza sativa*), Yang et al. (2008) found that CW PME activity and related content of demethylated pectin in the root tips were higher in the Al-sensitive cultivar than in the Al-resistant cultivar. This indicates that the higher density of polygalacturonic acid carboxylic groups in the CW causes a corresponding higher Al accumulation in the root tips and the CW. Also, transcriptional analysis of Al resistance in maize by Maron et al. (2008) revealed that Al upregulated the expression of the *PME* gene in both Al-resistant and Al-sensitive genotypes, while the level of upregulation of *PME* was higher in Al-sensitive genotypes. Furthermore, Horst et al. (1999) reported that short-term Al accumulation of roots was closely related to the pectin content in apical root sections of maize and faba bean (*Vicia faba*), and the binding of Al to the pectic matrix was closely positively correlated with Al-induced callose formation and thus Al sensitivity. Therefore, it appears that the binding of Al to pectins is closely related to Al sensitivity, since it was also reported that the Al-induced increase in pectin content of Al-sensitive cultivars was greater than that of Al-resistant cultivars (Eticha et al. 2005; Yang et al. 2008). Also in common bean, Rangel et al. (2009) found that the Al-induced root-growth inhibition was closely negatively related particularly to strongly bound CW Al. This suggests that the strong binding of Al to the pectic matrix of the CW is a main factor in Al toxicity rather than a resistance mechanism in common bean, although in earlier studies by Van et al. (1994), it was suggested that Al is detoxified by binding to pectins since the free carboxyl groups of pectin can bind or chelate Al^{3+} ions and cause cross-linking of pectin molecules (Klimashevskii and Dedov 1975).

In addition to pectins, the importance of hemicellulose in CW Al-binding capacity and thus Al toxicity has been suggested. By fractionating CW components, Yang et al. (2011) found that 75 % of the CW Al accumulated in the hemicellulose fraction 1 (HC1) compared to only 20 % in the CW pectin fraction. The interaction of Al with hemicellulose is not yet well understood, since according to the analysis of uronic acids in the different CW component fractions, the percentage of uronic acids in the pectin, HC1, and HC2 fractions were 72 %, 15 %, and 13 %, respectively. Xyloglucan is the most abundant hemicellulosic polysaccharide primary cell walls of dicotyledons. It functions by forming load-bearing cross-links among microfibrils, where they play a central role in modulating the mechanical properties of CWs (reviewed by Nishitani 1997). In Arabidopsis, Zhu et al. (2012) provided evidence that Al interacts specifically with xyloglucans. They postulated that the formation of an Al-xyloglucan complex inhibits cell wall loosening in the elongation zone of roots and thus contributes to inhibition of root elongation by Al.

The xyloglucan endotransglucosylase/hydrolases (XTHs) are enzymes that specifically use xyloglucan as a substrate and catalyze xyloglucan endotransglucosylase (XET) and/or xyloglucan endohydrolase (XEH) activities. They play a key role in the modification of CW structure and extensibility through the cleavage and re-formation of bonds between xyloglucan chains (Rose et al. 2002; Bray 2004). The XTH proteins are a large family of CW proteins which have 33 members known in the Arabidopsis genome (Rose et al. 2002; Bray 2004). In Arabidopsis, the expression of *XTH31* was suppressed by Al stress and thus has been suggested

to play a crucial role in the modulation of Al resistance through the regulation of the CW xyloglucan content and thus Al accumulation in roots (Zhu et al. 2012). Combination of the yeast two-hybrid assay and coimmunoprecipitation analysis revealed that XTH17 can interact with XTH31 in vitro (Zhu et al. 2014). These authors conclude that XTH17 and XTH31 may exist as a dimer at the plasma membrane conferring in vivo XET action, thus modulating CW Al-binding capacity and Al sensitivity of Arabidopsis. Further studies indicated that the O-acetylation of xyloglucan by the putative O-acetyltransferase TRICHOME BIREFRINGENCE-LIKE27 (TBL27 [AXY4]) affects Al sensitivity by modulation of Al-binding capacity in the hemicellulose xyloglucan (Zhu et al. 2014).

A decisive role of Al binding in the CW for Al toxicity is further supported by the studies of Xia et al. (2010) and Li et al. (2014) in rice. They characterized the plasma membrane-localized transporter, Nrat1 (Nramp aluminum transporter 1), belonging to the Nramp (natural resistance-associated macrophage protein) family specifically transporting the trivalent Al ion through the plasma membrane. The effective transport of Al from the apoplast to the symplast where it is ultimately sequestered in the vacuole plays an important role in the remarkably high Al tolerance of rice by reducing the level of toxic Al in the root CW.

4 Al Affects Cell Wall Properties

4.1 Cell Wall Extensibility

It has been demonstrated that Al treatment reduces root CW extensibility (Tabuchi and Matsumoto 2001; Ma et al. 2004). Through the analysis of CW components in root tips of both Al-resistant and Al-sensitive cultivars of wheat (*Triticum aestivum*), Tabuchi and Matsumoto (2001) and Zakir Hossain et al. (2006) showed that Al increased both the molecular mass of hemicellulosic polysaccharides and the amount of wall-bound ferulic acids particularly in the Al-sensitive cultivar. They speculated that phenolic acids may cross-link with other CW components such as hemicellulosic polysaccharides and thus induce the mechanical rigidity of the CW leading to the decrease in CW extensibility and inhibition of root elongation. Irrespective of whether pectin or hemicellulose is the primary binding site of Al in the CW, the binding of Al to either CW component will affect CW extension either directly physically or indirectly. Figure 5 schematically depicts the possible pathways how Al may affect the CW extensibility. For the indirect effect of CW Al, the replacement from the CW pectic matrix of Ca^{2+}, which plays a key role in controlling CW extensibility by the formation and cleavage of Ca bonds during cell elongation (Boyer 2009), or decreasing the effectiveness of CW-loosening enzymes, such as XTHs (Tabuchi and Matsumoto 2001; Ma et al. 2004; Wehr et al. 2004), and of cell wall structural proteins such as expansin (Cosgrove 1989) may be responsible. The studies in Arabidopsis (Yang et al. 2011) and bean

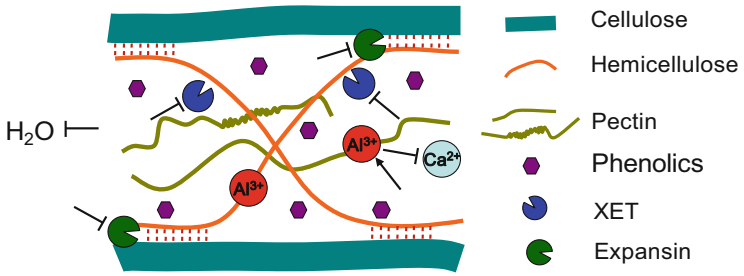

Fig. 5 A simplified model representing the effect of Al on cell wall (CW) extension. Under Al stress, Al^{3+} strongly binds to pectins and hemicellulose affecting the chemical and mechanical properties of the CW either directly and/or indirectly: (1) The Al cross-linked pectic and hemocellulosic cell wall matrix may lose its extensibility physically and/or physiologically by decreasing the activities of CW-loosening enzymes such as xyloglucan endotransglucosylase (XET). (2) Al rapidly and irreversibly displaces Ca^{2+} at the site of Ca^{2+}-pectate cross-linkages, which play a key role in controlling CW extensibility and thus cell elongation and development. Direct interaction of Al with CW structural proteins (expansin) may also affect CW extensibility. In addition, the Al-induced accumulation of phenolics involved in the cross-linking of structural CW components thus strengthening the CW wall may affect CWl porosity and limit water flow. Based on Yang et al. (2013)

(*Phaseolus vulgaris*) (Yang et al. 2012) have demonstrated that Al stress resulted in the inhibition of the expression of XTH genes and the activity of the CW-loosening enzyme XET in roots, which was related to the inhibition of root elongation by Al. In Arabidopsis roots, the reduction of the activity of this enzyme was accompanied with the deposition of callose an indicator of Al sensitivity (Yang et al. 2011). However, a direct interaction of Al^{3+} with structural CW proteins thus directly affecting CW extensibility cannot be ruled out.

The interaction of Al with CW components may also indirectly affect CW extension through the cell wall–plasma membrane–cytoskeleton continuum (Horst et al. 1999; Sivaguru et al. 2000). The interaction between CW and plasma membrane may be mediated by a cell wall-associated pectin receptor kinase (Kohorn and Kohorn 2012) which has been implicated in Al-induced root-growth inhibition in Arabidopsis (Sivaguru et al. 2003).

4.2 Cell Wall Porosity

The plant cell wall is a composite structure consisting of a cellulose–hemicellulose framework embedded within a matrix of pectins and proteins as mentioned above. The pores of the CW are the first barrier for mobile solutes such as ions, proteins, and water penetrating the wall (Brett and Waldron 1996), and plant cells interact with their environment through the porous network of the CW (Carpita et al. 1979). Generally, the pore diameter of the plant CW is in the range of 3.5–5.5 nm, which mainly depends on structure, hydrophobicity, chemical composition, and physical

properties of the CW (Carpita et al. 1979; Chesson et al. 1997). This porous structure of the matrix permits low-molecular-weight solutes to diffuse across the CW and interact with the plasma membrane, while for high-molecular-weight solutes the pore size impedes transport (Sattelmacher 2001). According to Baron-Epel et al. (1988), the pore size of the CW is mainly controlled by the pectic matrix. The Al-induced enhancement of the CW pectin content in the root tips and its cross-linking by Al through binding to the negatively charged sites (Horst et al. 2010) may affect the porosity of the wall. Whether binding of Al to hemicellulose through Al–xyloglucan interaction also affects root porosity needs further clarification.

Any change in the factors affecting the pectic matrix may change the porosity. For example, it was reported that low temperature decreased the pore size of the CW by modifying CW composition (Bauchot et al. 1999; Rajashekar and Lafta 1996). In general, dicots display a stronger response to B supply than monocots, which may be due to the higher B requirement of dicots. Enhanced Al toxicity in B-deficient dicot plant species (Stass et al. 2007) could also be related to the pore size of the cell wall which is affected by borate ester cross-linking of the pectic polysaccharide RG II (Fleischer et al. 1999). In a study on the interaction of Al toxicity and drought stress in common bean, Yang et al. (2010, 2011, 2013) presented circumstantial evidence that polyethylene glycol 6000 induces a rearrangement of the wall polymers and thus affects CW porosity. This restricted the penetration of Al^{3+} into the apoplast. Genes related to CW loosening or structure such as *XTH*, *beta-1,3-glucanase* (*BEG*), and *hydroxyproline-rich glycoprotein* (*HRGP*) appeared to play crucial roles (Fig. 6) in the PEG-induced modification of CW structure.

4.3 Blocking of Cell-to-Cell Trafficking by Callose

Cell wall deposited callose is a β-1,3-glucan with some β-1,6-branches and is produced by callose synthases and degraded by β-1,3-glucanases at the plasma membrane. Increased cytosolic Ca^{2+} ($[Ca^{2+}]_{cyt}$) and modification of the plasma membrane (PM) are crucial factors for the induction of callose synthesis by activating 1,3-β-glucan synthase (Kauss et al. 1990; Kauss 1996). Callose plays important roles during a variety of processes in plant development and/or in response to biotic and abiotic stresses including Al stress (Stass and Horst 2009; Chen and Kim 2009). Al-induced higher callose formation in the outer cortex cells of the DTZ compared with the cells of the MZ and EZ might be related to a higher PM depolarization in maize (Sivaguru et al. 1999) and higher $[Ca^{2+}]_{cyt}$ in the DTZ indicated by fluorescence resonance energy transfer-sensitized emission of the yellow cameleon 3.60 reporter in Arabidposis (Rincón-Zachary et al. 2010). Al-induced callose deposition in the root tips is positively correlated with Al-induced inhibition of root growth and Al accumulation in the root tips (Larsen et al. 1996; Horst et al. 1997; Yang et al. 2012) and proved to be a sensitive indicator of Al injury in roots (Stass and Horst 2009). Al-induced callose formation

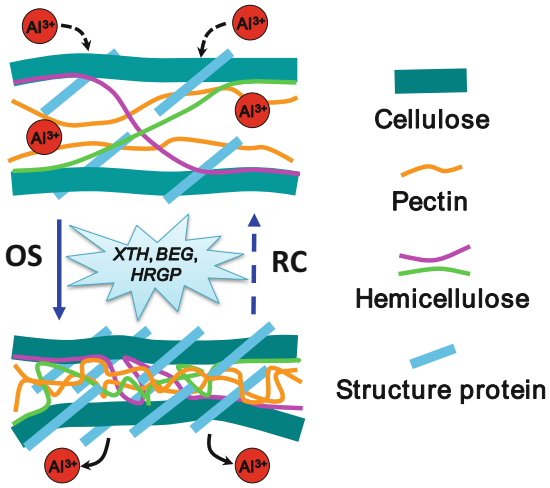

Fig. 6 A model representing the effect of osmotic stress (OS) on cell wall (CW) structure and Al binding, and the possible role of CW modification-related genes or structure proteins in the OS-induced change in CW porosity and thus Al binding to the CW in common bean plants. Under polyethylene glycol (PEG)-6000-induced OS, loss of water from the CW matrix leads to reduced CW porosity and excludes Al^{3+} from the apoplast, while the recovery of the CW from OS restores the pore size and allows Al^{3+} entry into the apoplast and its binding to the pectic matrix and/or hemicellulose. CW modification genes *xyloglucan endotransglucosylase/hydrolase* (*XTH*), *β-1,3-glucanase* (*BEG*) and the structural protein *HRGP* (*hydroxyproline-rich glycoprotein*) are supposed to be involved in the modification of CW porosity. Based on Yang et al. (2013)

has been successfully used as a reliable parameter for the classification of genotypes of different plant species for Al sensitivity (Wissemeier et al. 1992; Horst et al. 1997) and for the screening of maize cultivars for adaptation to acid Al-toxic soils (Collet and Horst 2001; Eticha et al. 2005; Narro and Arcos 2010).

Callose formation is not only an indicator of Al stress, but has also been implicated in Al toxicity. A recent study by Zhang et al. (2014) found that heterologous expression of the sweet sorghum (*Sorghum bicolor*) gene *SbGlu1*, which encodes a β-1,3-glucanase, reduced callose deposition and Al accumulation and enhanced the Al resistance in Arabidopsis. Callose can be deposited at plasmodesmata (PD) to regulate the cell-to-cell movement of molecules by controlling the size exclusion limit (SEL) of PD (Chen and Kim 2009). Microinjection of the dye lucifer yellow carbohydrazide into peripheral root cells of an Al-sensitive wheat cultivar (*Triticum aestivum*, cv Scout 66) before or after Al treatment revealed that the Al-induced inhibition of root growth resulted from the Al-induced blockage of cell-to-cell trafficking via the PD (Sivaguru et al. 2000). Further immunofluorescence combined with immunoelectron microscopic techniques using monoclonal antibodies against callose demonstrated that the Al-induced callose deposition at the PD is responsible for the blockage of the symplastic transport, which was further verified using a callose synthesis inhibitor. In addition, the expression of PD-associated proteins such as calreticulin and

unconventional myosin VIII was induced by Al and both proteins were co-localized with callose deposits. These results suggest that the extracellular Al-induced callose deposition at PD can effectively block symplastic transport and cell-to-cell signaling in higher plants.

5 Solute Flow

The rapid binding of Al in the root apoplast may reduce CW porosity and thus the mobility of particularly higher molecular solutes. This assumption has been confirmed by Schmohl and Horst (2000) who demonstrated a greatly Al-reduced release of acid phosphatase by maize suspension cells. However, these results can also be explained by a lower permeability of the plasma membrane for macromolecules. The study by Sivaguru et al. (2006) provided a more convincing evidence of the Al-induced inhibition of the apoplastic solute bypass flow in maize root apices by using the fluorescent probes HPTS (8-hydroxypyrene-1,3,6-trisulfonic acid, trisodium salt, molecular weight 524) and dextran-Texas Red conjugates (molecular weight 3000, 10,000, and 40,000) at the outer cortical cells, especially in the DTZ, and inhibition of transfer of these solutes to the xylem and finally the shoot. A contribution of Al-induced callose deposition to the inhibition of the apoplastic bypass flow could not be ruled out, since the inhibition of callose synthesis by pretreatment of the roots with 2-deoxy-D-glucose (DDG) prior to Al treatment partially alleviated the Al-induced inhibition of solute bypass flow.

It has been hypothesized that Al may not only affect the apoplastic flow of high-molecular solutes but also affect the root hydraulic conductivity (Kruger and Sucoff 1989; Maison and Bertsch 1997). Using artificial pectin membranes, Blamey et al. (1993) demonstrated that the binding of Al to pectin strongly reduced water permeability of the membranes in vitro. Gunsé et al. (1997) showed that Al decreased the hydraulic conductivity accompanied with reduced CW extensibility in an Al-sensitive maize cultivar. But Sivaguru et al. (2006) could not confirm an effect of Al on water flow from the roots to the shoot in maize.

However, Al may reduce the permeability of the plasma membrane for water and impede symplastic water transport, since Al strongly interacts also with membrane components affecting membrane structural properties such as fluidity and permeability (Vierstra and Haug 1978; Wagatsuma et al. 2005; Khan et al. 2009). In the root cortical cells of Northern red oak (*Quercus rubra* L.), Al decreased membrane permeability to water (Zhao et al. 1987; Chen et al. 1991) possibly by blocking aquaporins as suggested by gene expression analysis which suggested that Al suppressed the expression of genes coding for tonoplast aquaporins in rye (*Secale cereal* L.) (Milla et al. 2002). The role of Al on water transport is not yet well understood and urgently needs further experimental clarification given the importance of the Al/drought interaction for plant production on acid soils (Yang et al. 2013).

6 Conclusions

The apoplast of the most Al-sensitive apical root zone plays an important role in Al toxicity and resistance in plants. There is increasing evidence that inhibition of root growth is induced by Al directly and indirectly through interaction with CW structure and assembly mediated by phytohormones. An in-depth molecular characterization of hormone signaling regulating root growth plasticity via modification of cell wall properties in response to Al stress is urgently required and may represent a prerequisite for an improved understanding of general mechanisms of plant adaptation to a changing environment.

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