INTRODUCTION

The plant actin cytoskeleton plays a key role in the plant morphogenesis and is involved in polar cell growth, movement of subcellular organelles, cell division, and plant defense. Organization of actin cytoskeleton undergoes dynamic remodeling in response to internal developmental cues and diverse environmental signals. This dynamic behavior is regulated by numerous actin-binding proteins (ABPs) that integrate various signaling pathways. Production of the signaling lipids phosphatidic acid (PA) and phosphatidylinositol 4,5-bisphosphate affects the activity and subcellular distribution of several ABPs, and typically correlates with increased actin polymerization. Here we review current knowledge of the inter-regulatory dynamics between signaling phospholipids and the actin cytoskeleton in plant cells.

Keywords: actin, actin-binding proteins, capping protein, cytoskeleton, phosphatidic acid, phosphatidylinositol 4,5-bisphosphate, phospholipase D, signaling

UNIQUE STRUCTURAL PROPERTIES OF PIP2 AND PA DETERMINE THEIR BIOLOGICAL ACTIVITY

Although both PA and PIP2 are negatively charged (i.e., acidic) in the physiological pH range, they markedly differ in their structural and biophysical properties. PIP2 contains a bulky headgroup, with net charge ranging from –3 to –5 under physiological pH and an inverted conical shape that promotes positive curvature of membranes (Figure 1). Since total concentration of PIP2 in the plant plasma membrane is less than 1% (Munnik and Nielsen, 2011), PIP2 (together with other phosphoinositides, PIPs) is believed to function as an address label that defines membrane identity and as a landmark molecule for its protein partners, rather than having a general structural role in the lipid bilayer.

In contrast to the distinct structure of PIP2, that makes it very distinguishable in the membrane for its interaction partners, PA represents the simplest glycerophospholipid, consisting of a hydrophobic diacylglycerol (DAG) body and a single phosphate as the polar hydrophilic headgroup (Figure 1). PA is more abundant than PIP2 in the plant plasma membrane (usually between 5 and 10% of total phospholipids; Furt et al., 2010) and can change local properties of the lipid bilayer due to its cone-like shape, favoring negative membrane curvature (Kooijman et al., 2003; Tosterink and Munnik, 2011). Interestingly, the specificity of PA interactions with its binding proteins is the result of a unique PA property called the electrostatic/hydrogen bond switch, where the negative charge of the PA headgroup is increased from –1 to –2 and stabilized upon formation of hydrogen bonds with arginine and lysine residues of effector proteins (Kooijman et al., 2007).

In addition to differences in polar headgroups, distinct membrane properties of PIP2 and PA may also result from different acyl compositions. In tobacco leaves, PA is predominantly made of...
AtPI4P5K10–11 (Ischebeck et al., 2010). PI4P5Ks have an essential role in Arabidopsis (Brown and Auger, 2011). The key enzyme in PIP2 synthesis is phosphatidylinositol 4-phosphate 5-kinase (PI4P5K). In Arabidopsis, 11 genes encoding PI4P5K isoforms were identified (Mueller-Roeber and Pical, 2002). These genes could be further divided into two subgroups based on their overall structure, one group containing Arabidopsis PI4P5K1–9 and the other formed by PI4P5K10 and PI4P5K11 genes (Ischebeck et al., 2010). PI4P5Ks have an essential role in root-hair growth, pollen development, and guard cell opening (Munnik and Nielsen, 2011). Intriguingly, a double mutant of the fra10 mutant, coding for SAC-bearing PPI phosphatase Phospholipid signaling in actin dynamics

In addition to PPI formation, reduction in PPI levels is also likely to regulate the actin cytoskeleton. Phosphoinositide-specific phospholipase C (PI-PLC) is an enzyme that hydrolyzes PIP2 into DAG and inositol trisphosphate (IP3), and was shown to affect actin organization in Petunia pollen tubes by knockdown studies (Dowd et al., 2006). Moreover, two non-related families of phosphatases are present in plant genomes: inositol polyphosphate 5-phosphatases (5PTases), that can cleave both PIP2 and inositol polyphosphates, and PPI phosphatases containing SAC domain that preferentially cleave membrane PPIs. Interestingly, the fra7 mutant that has been identified as 5PTase15 implicated in controlling actin organization and secondary cell wall synthesis in fiber cells (Zhang et al., 2004). Actin disorganization was also shown in fra7 mutant, coding for SAC-bearing PPI phosphatase Phospholipid signaling in actin dynamics (Zhang et al., 2005).

**PA production**

In addition to ER-localized biosynthesis of PA that serves as a precursor for structural phospholipids and triacylglycerols, two distinct pathways can lead to formation of PA with signaling properties. The most studied pathway involves hydrolysis of structural phospholipids by phospholipase D (PLD), directly yielding PA. In comparison to yeast and animal genomes, the PLD family is expanded in plants with 12 genes in Arabidopsis and even more in other dicot and monocot genomes (Eliáš et al., 2002; Pleskot et al., 2012a). Interestingly, the PLDβ1 isoform from Arabidopsis and tobacco was found to interact directly with actin and is implicated in the regulation of actin polymerization (Kusner et al., 2003; Pleskot et al., 2010).

In addition to the PLD pathway, PA can be also produced by phosphorylation of DAG from the activity of diacylglycerol kinase (DGK). Intriguingly, “signaling” DAG in plant cells can be generated either from PIP2 via PI-PLC or from structural phospholipids via the activity of non-specific PLC (Munnik and Nielsen, 2011; Pukhlyahu et al., 2013), thus linking PPIs and PA signaling. The knowledge about plant DGKs is scarce and no molecular or genetic data are available that would support a role in actin regulation. However, several animal DGK isoforms have been implicated in actin regulation, and a plant DGK activity was found to be associated with F-actin in carrot cell cultures (Tan and Boss, 1992).

**MULTIFACETED ROLE OF PIP2 IN THE REGULATION OF ACTIN CYTOSKELETON**

There are several different ways that PIP2 can affect actin polymerization, dynamics, and association with the membrane: through direct binding and regulation of distinct ABPs, indirectly through regulation of the activity and localization of ROP (Rho of plants) GTPases, or via recruiting scaffolding proteins to the plasma membrane (Zhang et al., 2012).

Actin-binding proteins were among the first proteins whose biological activity was shown to be regulated by PIP2 (reviewed in Zhang et al., 2012). There seems to be a clear distinction between inhibiting and activating properties of PIP2 in

**Figure 1** Schematic representation of actin regulation by phosphatidylinositol 4,5-bisphosphate (PIP2) and phosphatidic acid (PA). Solid lines represent pathways leading to activation (arrow ends) or inhibition (arrow head). Dashed lines indicate pathways leading to PIP2/PA production/degradation. Dotted lines represent an induction of actin polymerization. A question mark indicates that experimental data are available only for non-plant cells. Enzymes generating PIP2 or PA are in green and proteins involved in phospholipid degradation or signaling attenuation are in red. In PIP2 and PA structural models, red and brown balls represent the oxygen and phosphorus atoms in headgroups, respectively, and carbon atoms are shown in cyan. ADF, actin-depolymerizing factor; CP, capping protein; Fractin, filamentous actin; DGK, diacylglycerol kinase; LPP, lipid phosphatase phosphatases; PP5K, phosphatidylinositol 4-phosphate 5-kinase; PI-PLC, phosphatidylinositol-specific phospholipase C; PLD, phospholipase D; ROP, Rho of plants.
Arabidopsis contains five ABPs including members of ADF (actin-depolymerizing factor) protein families (Saarikangas et al., 2010), in vinculin, talin, spectrin, ERM (ezrin/radixin/moesin), and villin have been described to be regulated by PIP2 to date in plants, four distinct ABP classes (profilin, ADF/cofilin, CP, and villin) have been described to be regulated by PIP2 to date (reviewed in Day et al., 2011). Profilin colocalizes with PIP2 at plant profilin does not catalyze nucleotide exchange on actin and Blanchoin, 2006). In contrast to non-plant counterparts, plant profilin directly binds PIP2 (Kovar et al., 2001) and it could be speculated that similar to its animal homologs, profilin can then dissociate from profilin-actin complexes releasing free G-actin (Witke, 2004). Interestingly, plant profilin also inhibits spontaneous nucleation of actin and prevents assembly at the slow-growing, pointed end of actin filaments (Staiger et al., 2006). In contrast to non-plant counterparts, plant profilin directly binds PIP2 at the tip of growing root hairs (Braun et al., 1999). Moreover, plant profilin directly binds PIP2 (Kovar et al., 2001) and it could be speculated that similar to its animal homologs, profilin can then dissociate from profilin-G-actin complexes releasing free G-actin (Witke, 2004). Interestingly, plant profilin also inhibits the activity of PIP2-degrading enzyme, PI-PLC (Kovar et al., 2001).

Proteins of the ADF/cofilin family represent conserved ABPs across eukaryotes (Hussey et al., 2002). ADF/cofilin recycles actin monomers by severing and creating new filament ends (Andrianantoandro and Pollard, 2006; Hentz et al., 2011). Zea mays (Zm) ADF3 directly binds and is inhibited by PIP2. Moreover, similar to the profilin-PIP2 interaction, the ZmADEF binding of PIP2 suppresses the activity of PI-PLC (Gingabissou et al., 1998). Similar findings were reported for ADF1 from lily pollen (Alwood et al., 2002), suggesting that PPI regulation is a common feature of plant ADF/cofilin isoforms.

Villin belongs to the ABP protein superfAMILY GelSolin/Villin/Fragmin and is composed of six gelosin-homology domains at its core and a villin headpiece domain at its C-terminus. Arabidopsis contains five VILIN genes, however, genes coding for gelosin and fragmin are not present in model plant genomes. Interestingly, actin-severing activity of ABP29, a probable splice variant of the 135-kDa villin from lily, was shown to be inhibited by PIP2 (Xiang et al., 2007). However, the analogous regulation of full-length plant villin remains to be demonstrated.

Capping protein is a heterodimeric protein distributed across almost all eukaryotes (Pleskot et al., 2012b), it binds to the fast growing end of actin filaments, thus inhibiting polymerization. CP bound to actin filaments also protects against disassembly (Huang et al., 2003). Similar to animal cells, it was shown that the ability of Arabidopsis CP to bind actin fast-growing ends is inhibited PIP2 in vitro (Huang et al., 2006). However, unlike animal and yeast CPs, the Arabidopsis CP homolog has been also identified as a direct target of PA both in vitro and in vivo (see below for more details; Huang et al., 2006; Li et al., 2012a).

Rho of plants small GTPases are a plant-specific subfamily and sole members of the Rho/Rac/Cdc42 family of Ras-related G-proteins in plants, where they serve as "master switches" involved in diverse signaling and developmental pathways. Activated ROP variants are associated with the plasma membrane, where they are thought to control cell growth by coordinating actin organization and membrane trafficking (Michal et al., 2011). Importantly, PIP2 was shown to colocalize with ROP GTPases at the apical plasma membrane of tobacco pollen tube and pollen ROP physically interacts with PH domain activity (Kost et al., 1999; Yakovsky et al., 2008). Importantly, type II plant ROP GTPases have a polybasic motif at the C-terminal part of the protein, which is necessary for plasma membrane localization (Lavy and Yakovsky, 2006). It is therefore tempting to speculate that this polybasic motif binds PIP2, directly, as described for many members of the human small GTPase family (Heo et al., 2006). Furthermore, it was recently shown that PIP4P5K regulates actin dynamics in pollen tubes by counteracting Rho-GDI (Rho-guanine nucleotide dissociation inhibitor), thereby regulating the pool of membrane-localized ROP GTPases (Isebebeck et al., 2011).

**PA REGULATES PLANT ACTIN CYTOSKELETON DYNAMICS THROUGH CP**

In the last decade, several studies describe changes in signaling PA levels that generate a pronounced effect on plant actin cytoskeleton organization (Lee et al., 2013; Motes et al., 2005; Huang et al., 2006; Apostolakos et al., 2008; Pleskot et al., 2010, 2012a). Given the profound effect of PA production on actin polymerization in eukaryotes, it is surprising that no ABPs regulated by PA were described in animal or yeast cells. Indeed, the PA effect on actin in animals appears to be mainly indirect, by controlling production of PIP2 through PI4P5Ks [(Roach et al., 2012); and see below]. In plant cells on the other hand, CP was found to be regulated by PA as well as PPIs in vitro (Huang et al., 2006). Furthermore, the critical role of PA in plant CP regulation was confirmed by utilizing cp knockdown mutants (Li et al., 2012a,b). Structural aspects of the ACP inhibition by PA highlight a key role for the C-terminal part of CPA subunit, as demonstrated through molecular dynamics simulations (Pleskot et al., 2012b). The fact that a direct interaction between and PLDP exists in plant cells (Pleskot et al., 2010) leads to the hypothesis of a positive feedback loop model for actin dynamics regulation by PLDP and PA. Briefly, intracellular or intercellular signals cause activation of PLDP and subsequently increase the local PA concentration. PA binds CP and prevents its binding to the fast growing end of actin filaments, thus promoting actin polymerization. Newly formed actin filaments promote PLDP activity, leading to local enhancement of PA concentration
and further enhancement of actin assembly (Plesek et al., 2010, 2013).

CONCLUDING REMARKS AND HYPOTHESES

During the last 20 years, multiple direct and indirect interactions between PI(4,5)P2 and PA-mediated signaling pathways and the regulation of actin dynamics have been revealed. Despite the fact that the regulation of actin dynamics is a point of convergence for many signaling pathways and exhibits complex feedback regulation (Figure 1), general conclusions can be drawn. The elevation of PI(4,5)P2 and PA levels increases both density and complexity of the actin network and conversely the inhibition of PA/PIP2 production leads to actin filament disruption. Although many similarities can be found in ARK-phospholipid regulation between plant and animal cells, there is one principal difference: in plants, PIP2 levels are 10 times lower than PA levels (Drobak, 1993; Zonia and Munek, 2004). It is therefore tempting to speculate that many plant ABPs adapted to the distinct levels of PA and PIP2. It might be expected that additional ABPs interact with PA and/or PIP2 in plant cells, and this should be a topic for future exploration.

Many published reports on ABP-phospholipid regulation assume that the protein–lipid bivalent interaction is mono-specific, i.e., a single species of lipid is responsible for recruiting a given ABP to the membrane. However, work from animal and yeast cells has shown that a mono-specific reaction is the exception rather than the rule: for the majority of lipid effectors, membrane translocation probably depends both on a specific lipid but also on the surrounding lipid environment (Moravcevic et al., 2012). Indeed, several recent computational studies, albeit not on proteins involved in the regulation of the actin dynamics, show the involvement of other phospholipids for protein domains previously thought to function in a mono-specific way. Koosjman et al. (2007) experimentally described the positive effect of phosphatidylethanolamine on the PA binding by APD1K1, ATCTR1, and Raf-1. Similar results were obtained for the binding of PPIs by PH, PX, and FYVE domains (Pachoudia and Sanseim, 2008, 2009; Lamb et al., 2011). Several PA-binding proteins also have affinity for different PPIs. The binding of another signaling phospholipid could be mediated by the same domain, as in the case of APD1K1 and p47Phox PA domain, or through a completely distinct domain, for example the C1 and C2 domains of mammalian PKCε (Testerink and Munnek, 2011), but the molecular details are largely missing. Interestingly, the C1-domain, a canonical DAG-binding motif, binds more strongly to DAG embedded in the negatively charged membrane and DAG-mediated targeting of effector proteins thus seems to be also enhanced by synergistic binding to acidic phospholipids, such as PA and PIP2 (Colón-Contreras and Kazanjian, 2006). From this point of view, dual regulation of plant CP by both PA and PIP2 (Huang et al., 2006) might represent just the tip of an iceberg. A cooperative effect between PA and PIP2 in the regulation of the actin dynamics could be also indirect. Recently, Roach et al. (2012) described the ability of PA to activate PI4P5K and the authors showed the crucial importance of membrane targeting of PI4P5K by PA in the regulation of actin reorganization in animal cells. The activation of kinase activity by PA was shown for PI4P5K1 (Perera et al., 2003). Given the fact that several PLD isoforms are activated by PIP2 (Li et al., 2009), one can expect that a vivid crosstalk between PA and PIP2 signaling to the actin cytoskeleton exists in all eukaryotic cells.

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