Viewpoints

Disrupted actin: a novel player in pathogen attack sensing?

Summary

The actin cytoskeleton is widely involved in plant immune responses. The majority of studies show that chemical disruption of the actin cytoskeleton increases plant susceptibility to pathogen infection. Similarly, several pathogens have adopted this as a virulence strategy and produce effectors that affect cytoskeleton integrity. Such effectors either exhibit actin-depolymerizing activity themselves or prevent actin polymerization. Is it thus possible for plants to recognize the actin's status and launch a counterattack? Recently we showed that chemical depolymerization of actin filaments can trigger resistance to further infection via the specific activation of salicylic acid (SA) signalling. This is accompanied by several defence-related, but SA-independent, effects (e.g. callose deposition, gene expression), relying on vesicular trafficking and phospholipid metabolism. These data suggest that the role of actin in plant–pathogen interactions is more complex than previously believed. It raises the question of whether plants have evolved a mechanism of sensing pathological actin disruption that eventually triggers defence responses. If so, what is the molecular basis of it? Otherwise, why does actin depolymerization specifically influence SA content but not any other phytohormone? Here we propose an updated model of actin's role in plant–microbe interactions and suggest some future directions of research to be conducted in this area.

Actin's involvement in plant immunity is being broadly studied (Li & Day, 2019). Plant immunity consists of two layers, separated temporally and spatially. The first one is associated with the plasma membrane and is dependent on the recognition of conserved molecules called microbe-associated molecular patterns (MAMPs) or damage-associated molecular patterns (DAMPs) by pattern-recognizing receptors (PRRs). This immune layer is entitled pattern-triggered immunity (PTI). The second layer, effector triggered immunity (ETI), is based on the intracellular recognition of effectors, molecules secreted by pathogens inside cells to inhibit immune responses (Jones & Dangl, 2006).

The actin cytoskeleton is a highly dynamic structure maintained by a balance between monomeric G-actin and polymerized filamentous F-actin. The filaments are constantly growing from one barbed end and shortening from the other one (Porter & Day, 2016). The speed and direction of their growth are regulated by numerous factors (e.g. actin depolymerizing factors (ADFs)). In their resting state, actin filaments maintain cellular metabolism, providing a 'signalling friendly' environment, that is, by the correct recycling of the PRRs. Shortly after sensing a pathogen, actin is reorganized and its density increases at the infection site (Porter & Day, 2016). In this case, actin filaments serve as a delivery pathway for compounds restricting microbial spread (e.g. callose) and sending DAMPs (e.g. Pep1, oligogalacturonides) to the neighbouring cells (Choi & Klessig, 2016) (Fig. 1a). Actin reorganization can be triggered not only by living pathogens, but also by treatment with MAMPs (e.g. flg22, elf18, chitin) or DAMPs. The MAMP-induced remodelling requires reactive oxygen species (ROS) generated by RBOHD, defence-associated NADPH oxidase (Li *et al.*, 2017). If such polymerization is prevented/blocked in the presence of drugs (such as latrunculin B or cytochalasin E) or by genetically affected ADFs, several downstream events fail, thus leading to a higher susceptibility to pathogens (Henty-Ridilla *et al.*, 2013; Badet *et al.*, 2017; Li & Day, 2019). This brings the general conclusion that actin depolymerization causes susceptibility (Fig. 1b).

Several actin remodelling factors were also described in the context of immunity. For example, ADF4 in *Arabidopsis thaliana* is connected with the R-protein RPS5 recognizing the *Pseudomonas syringae* effector AvrPphB (Henty-Ridilla *et al.*, 2013, 2014; Li *et al.*, 2017). In wheat, ADF3 negatively regulates resistance to *Puccinia striiformis* f. sp. *tritici* in ROS-dependent manner (Tang *et al.*, 2015). Profilins form a complex with several other proteins to facilitate actin assembly and also binding membrane phospholipids (Pernier *et al.*, 2016). The transcription level of *AtPRF3* in *A. thaliana* decreased upon flg22 treatment, but the *A. thaliana prf3* mutant shows a stronger response to flg22 in actin density, ROS burst and root growth assay. Interestingly, this is followed by increased susceptibility to *P. syringae* (Sun *et al.*, 2018). Formins, particularly FORMIN4, contribute to local actin dynamics during interaction between *A. thaliana* and the nonhost pathogen *Blumeria graminis*, especially to the formation of cell wall appositions, the first line of plant defence composed of callose, proteins and phenolic compounds (Sassmann *et al.*, 2018). Capping protein (CP) is a major regulator of actin dynamics. For actin polymerization to occur, its expression needs to be downregulated, while its constitutive deficiency in the *A. thaliana cpb1* mutant results in a high density of filaments (Li *et al.*, 2015). However, the *cpb1* mutant supported higher bacterial growth, while CP-overexpressing plants were more resistant to *P. syringae* infection compared with the wild-type (WT) (Li *et al.*, 2017). Interestingly, to induce actin polymerization, CP needs to be downregulated after binding the phosphatidic acid (PA) that is derived from the activity of phospholipase D (Pleskot *et al.*, 2010; Li *et al.*, 2015). This launches a positive feedback loop, as F-actin activates phospholipase D of *Nicotiana tabacum*, while G-actin

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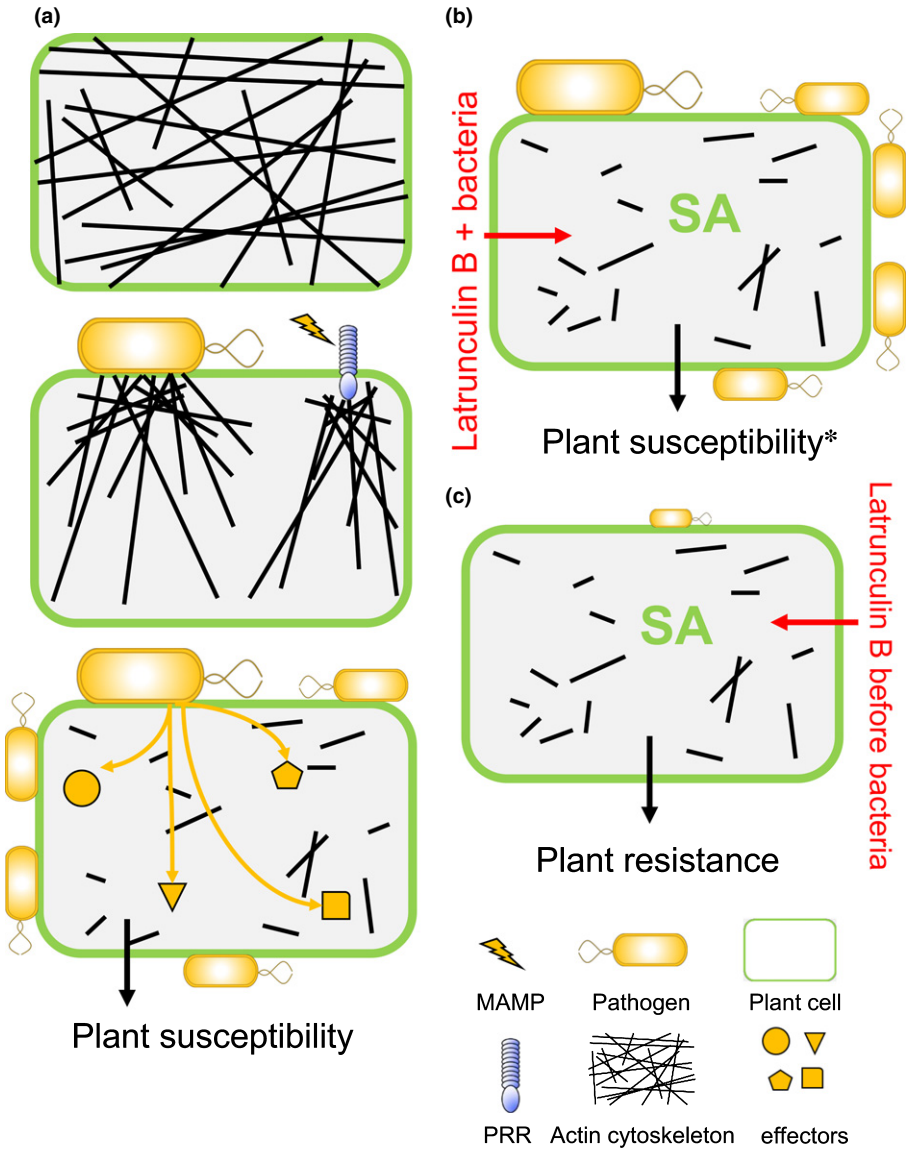

Fig. 1 (a) Model of the role of actin dynamics in plant–pathogen interactions showing the stages from normal actin cytoskeleton through reorganization after pathogen recognition to effects of secreted actin-depolymerizing effectors that lead to plant susceptibility. (b, c) Two distinct scenarios upon actin depolymerization after treatment with latrunculin B: (b) latrunculin B treatment leads to plant susceptibility; (c) latrunculin B pretreatment leads to induced resistance. MAMP, microbe-associated molecular pattern; PRR, pattern-recognizing receptor; SA, salicylic acid pathway. *, or no effect.

inhibits it *in vitro* (Pleskot *et al.*, 2010). This fits together with the fact that a PA increase follows the recognition of MAMPs and DAMPs, but also the application of salicylic acid (SA) (Kalachova *et al.*, 2013; Cacas *et al.*, 2017; Gully *et al.*, 2019). At the same time, chemical disruption of actin also triggers SA accumulation and SA-responsive genes (Matouskova *et al.*, 2014; Kalachova *et al.*, 2019; Leontovycova *et al.*, 2019). An activated SA pathway by actin disruption could even lead to increased plant resistance to pathogens (Leontovycova *et al.*, 2019) (Fig. 1c). This places actin remodelling at a crossroads of different signalling cascades. Another important connection of actin filaments with immunity lies in organelle movement coordination during pathogen attack. For example, actin is required for the anchoring of stromules, connecting the chloroplast and nucleus during infection (Kumar *et al.*, 2018). Chloroplasts are the major site of SA biosynthesis, so it is no surprise that pathogens may target chloroplasts to suppress SA-dependent immunity. This was recently described for bacterial and viral effectors (Medina-Puche *et al.*, 2019) and suggests an existing pathway, directly linking the plasma membrane to chloroplasts and activating plant defence. We believe that the actin network could be such a pathway.

Indeed, several pathogen effectors target the actin cytoskeleton's integrity to suppress plant immune responses. These effectors exhibit either actin-depolymerizing activity themselves or prevent actin polymerization. The *P. syringae*-secreted effector HopW1 disrupts the actin cytoskeleton and interacts with isoform 7 of vegetative actin (ACT7) (Jelenska *et al.*, 2014; Kang *et al.*, 2014). The Columbia ecotype of *A. thaliana* shows susceptibility when infected with *P. syringae* possessing this effector. However, in the Wassilewskija ecotype (Ws), HopW1 is recognized by WIN2 and WIN3 proteins with the subsequent onset of defence pathways triggering resistance against bacteria in Ws (Jelenska *et al.*, 2014). Another effector, HopG1, is responsible for the induction of cytoskeletal reorganization and infection-associated chlorosis. HopG1 interacts indirectly with actin filaments via forming a complex with mitochondria-localized kinesin protein. Moreover, the T3SS-deficient *P. syringae* strain *ΔhrpH* is both avirulent and unable to trigger the second phase of actin remodelling during PTI

in *A. thaliana* (Shimono *et al.*, 2016). It is worth noting that microtubules (MTs), the second component of the plant cytoskeleton, are also affected by secreted effectors, HopE1 (Cheong *et al.*, 2014) and XopL (Erickson *et al.*, 2018). MT disruption causes an increase in susceptibility to pathogens, as was shown for actin disruption (Schmidt & Panstruga, 2007; Lee *et al.*, 2012). However, treatment of *A. thaliana* with oryzalin, an MT-depolymerizing drug, does not trigger a strong immune response connected to SA (Matoušková *et al.*, 2014).

As actin polymerization occurs during PTI, and pathogens try to overcome it by secreting effectors leading to actin depolymerization, is it possible that plants have evolved another mechanism to sense a 'disrupted actin state' and relaunch immunity? If so, what mechanism lies beyond, and what molecule(s) is perceived? Is it based on a receptor-like signalling (e.g. free G-actin, short F-actin or ADFs interact with a receptor and this launches plant immunity) or is it an indirect connection (e.g. through the disturbed vesicular trafficking or phytohormones)? For example, actin was already shown to be a DAMP for mammalian (Srinivasan *et al.*, 2016) and insect (Ahrens *et al.*, 2012) cells.

We believe that future research can benefit hugely from natural variation studies. Indeed, a genome-wide associated study (GWAS) based on the screening of susceptibility to *Sclerotinia sclerotiorum* has already highlighted ARPC4, a new player involved in quantitative disease resistance and an actin organization regulator, both in the resting state and in response to infection (Badet *et al.*, 2019). Forward genetics can also be fruitful. In this case, it might be of interest to perform the ethyl methanesulfonate mutagenesis of plants with labelled actin filaments or with proteins responding to actin disruption (e.g. *PR2*). *PR2* expression could be a good marker, as it is induced after the chemical disruption of actin independently of SA (Kalachova *et al.*, 2019), so an impaired SA pathway in mutants will not influence the result. Reverse genetics is useful to understand signalling, but it needs to be carefully applied while working with immunity-related mutants. Indeed, many plants with altered immune pathways exhibit strong pleiotropic phenotypes, including growth retardation (Rate *et al.*, 1999; Yi & Richards, 2008). The behaviour of those plants is thus significantly affected by hormonal misregulation, especially SA increase (Pluharova *et al.*, 2019). The crossing of those mutants will bring new biases that need to be carefully interpreted. All this significantly limits actin-remodelling research, as we cannot fully exclude the pleiotropic effects of actin disruption caused by a pharmacological approach, while null mutants make time-shift studies impossible. The creation of transgenic plants with inducible actin depolymerization would therefore be very useful. One such line was recently prepared for animal studies (Harterink *et al.*, 2017) and has already been applied in plants as part of research into actin remodelling in lateral root formation (Vilches Barro *et al.*, 2019).

The specificity of triggering an SA pathway by actin disruption encourages us to believe that there is a unique signalling pathway involved. This pathway will probably overlap with other cell responses that coincide with cytoskeleton degradation (such as autophagy, senescence of induced cell death), but will include strict feedback regulation to prevent irreversible effects. Much still remains to be described. What is responsible for triggering the SA pathway? We can hypothesize about the earlier-mentioned receptor-like recognition, but it could also be that some SA-binding proteins exhibit actin-binding properties. In such a case, this double binding (or its interruption) could lead to further induction of an SA pathway. In human cells, actin ACTN1 and actin-binding filamin B (FLNB), the proteins directly involved in cytoskeletal rearrangements, were found to be SA-binding proteins (Choi *et al.*, 2019). In plants, the SA-binding proteins are mostly associated with respiration, photosynthesis, or signalling; no direct interactions with the cytoskeleton have yet been reported (Manohar *et al.*, 2014; Pokotylo *et al.*, 2019).

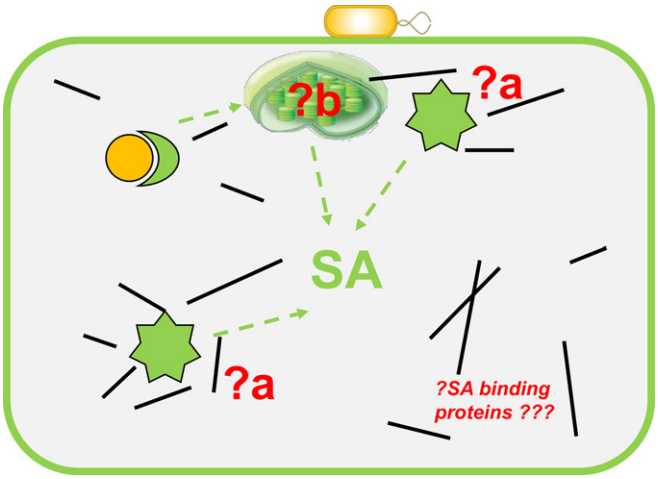

Fig. 2 How does actin depolymerization activate a salicylic acid (SA) pathway? In plants there is a receptor for depolymerized actin filaments (direct effect) (a). Depolymerized actin affects any of the processes such as organelle movements that subsequently activate an SA pathway (indirect effect) (b). Increased SA concentration is mediated through the effects of actin on SA-binding proteins.

Is the triggering of plant immunity (the SA pathway, in particular) caused by the disruption of actin filaments conserved during the evolutionary process? The SA pathway is conserved in land plants (Wang *et al.*, 2015). Two actin genes have already been seen in algae, while *A. thaliana* plants possess 12 isoforms of actin genes, expressed specifically in different tissue types and specific developmental stages (Slajcherova *et al.*, 2012). Similarly, CP-regulating actin polymerization has conserved sequencing found among all eucaryots (Cooper & Sept, 2008). This suggests that the mechanism of actin disruption sensing will be conserved across all land plants too. This may be investigated in parallel in diverse model systems.

Recent studies have found some answers, but they have also added more complexity to the current model of the role of the actin cytoskeleton in plant–microbe interactions (Fig. 2). Here we propose that a disrupted actin state be considered a plant cell component, which is sensed by a specific mechanism and thus triggers immune-like responses.

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Author contributions

HL, TK and MJ conceived the study; HL, TK and MJ wrote the manuscript; MJ drew the figures; and all authors commented and approved the text.

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References

Ahrens S, Zelenay S, Sancho D, Hanč P, Kjær S, Feest C, Fletcher G, Durkin C, Postigo A, Skehel M *et al.* 2012. F-actin is an evolutionarily conserved damage-associated molecular pattern recognized by DNGR-1, a receptor for dead cells. *Immunity* **36**: 635–645.

Badet T, Leger O, Barascud M, Voisin D, Sadon P, Vincent R, Le Ru A, Balague C, Roby D, Raffaele S. 2019. Expression polymorphism at the *ARPC4* locus links the actin cytoskeleton with quantitative disease resistance to *Sclerotinia sclerotiorum* in *Arabidopsis thaliana*. *New Phytologist* **222**: 480–496.

Badet T, Peyraud R, Mbengue M, Navaud O, Derbyshire M, Oliver RP, Barbacci A, Raffaele S. 2017. Codon optimization underpins generalist parasitism in fungi. *eLife* **6**: 22472.

Cacas JL, Gerbeau-Pissot P, Fromentin J, Cantrel C, Thomas D, Jeannette E, Kalachova T, Mongrand S, Simon-Plas F, Ruelland E. 2017. Diacylglycerol kinases activate tobacco NADPH oxidase-dependent oxidative burst in response to cryptogein. *Plant, Cell & Environment* **40**: 585–598.

Cheong MS, Kirik A, Kim JG, Frame K, Kirik V, Mudgett MB. 2014. AvrBsT acetylates Arabidopsis ACIP1, a protein that associates with microtubules and is required for immunity. *PLoS Pathogens* **10**: e1003952.

Choi HW, Klessig DF. 2016. DAMPs, MAMPs, and NAMPs in plant innate immunity. *BMC Plant Biology* **16**: 232.

Choi HW, Wang L, Powell AF, Strickler SR, Wang D, Dempsey DA, Schroeder FC, Klessig DF. 2019. A genome-wide screen for human salicylic acid (SA)-binding proteins reveals targets through which SA may influence development of various diseases. *Scientific Reports* **9**: 13084.

Cooper JA, Sept D. 2008. New insights into mechanism and regulation of actin capping protein. *International Review of Cell and Molecular Biology* **267**: 183–206.

Erickson JL, Adlung N, Lampe C, Bonas U, Schattat MH. 2018. The *Xanthomonas* effector XopL uncovers the role of microtubules in stromule extension and dynamics in *Nicotiana benthamiana*. *Plant Journal* **93**: 856–870.

Gully K, Pelletier S, Guillou MC, Ferrand M, Aligon S, Pokotylo I, Perrin A, Vergne E, Fagard M, Ruelland E *et al.* 2019. The SCOOP12 peptide regulates defense response and root elongation in *Arabidopsis thaliana*. *Journal of Experimental Botany* **70**: 1349–1365.

Harterink M, da Silva ME, Will L, Turan J, Ibrahim A, Lang AE, van Battum EY, Pasterkamp RJ, Kapitein LC, Kudryashov D *et al.* 2017. DeActs: genetically encoded tools for perturbing the actin cytoskeleton in single cells. *Nature Methods* **14**: 479–482.

Henty-Ridilla JL, Li J, Day B, Staiger CJ. 2014. ACTIN DEPOLYMERIZING FACTOR4 regulates actin dynamics during innate immune signaling in Arabidopsis. *Plant Cell* **26**: 340–352.

Henty-Ridilla JL, Shimono M, Li J, Chang JH, Day B, Staiger CJ. 2013. The plant actin cytoskeleton responds to signals from microbe-associated molecular patterns. *PLoS Pathogens* **9**: e1003290.

Jelenska J, Kang Y, Greenberg JT. 2014. Plant pathogenic bacteria target the actin microfilament network involved in the trafficking of disease defense components. *Bioarchitecture* **4**: 149–153.

Jones JD, Dangl JL. 2006. The plant immune system. *Nature* **444**: 323–329.

Kalachova T, Iakovenko O, Kretinin S, Kravets V. 2013. Involvement of phospholipase D and NADPH-oxidase in salicylic acid signaling cascade. *Plant Physiology and Biochemistry* **66**: 127–133.

Kalachova T, Janda M, Šašek V, Ortmannová J, Nováková P, Dobrev IP, Kravets V, Guivarc'h A, Moura D, Burketová L *et al.* 2019. Identification of salicylic acid-independent responses in an Arabidopsis phosphatidylinositol 4-kinase beta double mutant. *Annals of Botany* **125**:775–784.

Kang Y, Jelenska J, Cecchini NM, Li Y, Lee MW, Kovar DR, Greenberg JT. 2014. HopW1 from *Pseudomonas syringae* disrupts the actin cytoskeleton to promote virulence in Arabidopsis. *PLoS Pathogens* **10**: e1004232.

Kumar AS, Park E, Nedo A, Alqarni A, Ren L, Hoban K, Modla S, McDonald JH, Kambhamettu C, Dinesh-Kumar SP *et al.* 2018. Stromule extension along microtubules coordinated with actin-mediated anchoring guides perinuclear chloroplast movement during innate immunity. *eLife* **7**: e23625.

Lee AH, Hurley B, Felsensteiner C, Yea C, Ckurshumova W, Bartetzko V, Wang PW, Quach V, Lewis JD, Liu YC *et al.* 2012. A bacterial acetyltransferase destroys plant microtubule networks and blocks secretion. *PLoS Pathogens* **8**: e1002523.

Leontovycova H, Kalachova T, Trda L, Pospichalova R, Lamparova L, Dobrev PI, Malinska K, Burketova L, Valentova O, Janda M. 2019. Actin depolymerization is able to increase plant resistance against pathogens via activation of salicylic acid signalling pathway. *Scientific Reports* **9**: 10397.

Li J, Cao L, Staiger CJ. 2017. Capping protein modulates actin remodeling in response to reactive oxygen species during plant innate immunity. *Plant Physiology* **173**: 1125–1136.

Li J, Henty-Ridilla JL, Staiger BH, Day B, Staiger CJ. 2015. Capping protein integrates multiple MAMP signalling pathways to modulate actin dynamics during plant innate immunity. *Nature Communications* **6**: 7206.

Li P, Day B. 2019. Battlefield cytoskeleton: turning the tide on plant immunity. *Molecular Plant–Microbe Interactions* **32**: 25–34.

Manohar M, Tian M, Moreau M, Park SW, Choi HW, Fei Z, Friso G, Asif M, Manosalva P, von Dahl CC *et al.* 2014. Identification of multiple salicylic acid-binding proteins using two high throughput screens. *Frontiers in Plant Science* **5**: 777.

Matouskova J, Janda M, Fiser R, Sasek V, Kocourkova D, Burketova L, Duskova J, Martinec J, Valentova O. 2014. Changes in actin dynamics are involved in salicylic acid signaling pathway. *Plant Science* **223**: 36–44.

Medina-Puche L, Tan H, Dogra V, Wu M, Rosas-Diaz T, Wang L, Ding X, Zhang D, Fu X, Kim C *et al.* 2019. A novel pathway linking plasma membrane and chloroplasts is co-opted by pathogens to suppress salicylic acid-dependent defences. *BioRxiv*. doi: 10.1101/837955.

Pernier J, Shekhar S, Jegou A, Guichard B, Carlier MF. 2016. Profilin interaction with actin filament barbed end controls dynamic instability, capping, branching, and motility. *Developmental Cell* **36**: 201–214.

Pleskot R, Potocky M, Pejchar P, Linek J, Bezvoda R, Martinec J, Valentova O, Novotna Z, Zarsky V. 2010. Mutual regulation of plant phospholipase D and the actin cytoskeleton. *The Plant Journal* **62**: 494–507.

Pluharova K, Leontovycova H, Stoudkova V, Pospichalova R, Marsik P, Kloucek P, Starodubtseva A, Iakovenko O, Krckova Z, Valentova O *et al.* 2019. "Salicylic acid mutant collection" as a tool to explore the role of salicylic acid in regulation of plant growth under a changing environment. *International Journal of Molecular Sciences* **20**: 6365.

Pokotylo I, Kravets V, Ruelland E. 2019. Salicylic acid binding proteins (SABPs): the hidden forefront of salicylic acid signalling. *International Journal of Molecular Sciences* **20**: 4377.

Porter K, Day B. 2016. From filaments to function: the role of the plant actin cytoskeleton in pathogen perception, signaling and immunity. *Journal of Integrative Plant Biology* **58**: 299–311.

Rate DN, Cuenca JV, Bowman GR, Guttman DS, Greenberg JT. 1999. The gain-of-function Arabidopsis *acd6* mutant reveals novel regulation and function of the salicylic acid signaling pathway in controlling cell death, defenses, and cell growth. *The Plant Cell* **11**: 1695–1708.

Sassmann S, Rodrigues C, Milne SW, Nenninger A, Allwood E, Littlejohn GR, Talbot NJ, Soeller C, Davies B, Hussey PJ *et al.* 2018. An immune-responsive cytoskeletal-plasma membrane feedback loop in plants. *Current Biology* **28**: 2136–2144.e7.

Schmidt SM, Panstruga R. 2007. Cytoskeleton functions in plant–microbe interactions. *Physiological and Molecular Plant Pathology* **71**: 135–148.

Shimono M, Lu YJ, Porter K, Kvitko BH, Henty-Ridilla J, Creason A, He SY, Chang JH, Staiger CJ, Day B. 2016. The *Pseudomonas syringae* type III Effector HopG1 induces actin remodeling to promote symptom development and susceptibility during infection. *Plant Physiology* **171**: 2239–2255.

Slajcherova K, Fiserova J, Fischer L, Schwarzerova K. 2012. Multiple actin isotypes in plants: diverse genes for diverse roles? *Frontiers in Plant Science* **3**: 226.

Srinivasan N, Gordon O, Ahrens S, Franz A, Deddouche S, Chakravarty P, Phillips D, Yunus AA, Rosen MK, Valente RS *et al.* 2016. Actin is an evolutionarily-conserved damage-associated molecular pattern that signals tissue injury in Drosophila melanogaster. *eLife* **5**: e19662.

Sun H, Qiao Z, Chua KP, Tursic A, Liu X, Gao YG, Mu Y, Hou X, Miao Y. 2018. Profilin negatively regulates formin-mediated actin assembly to modulate PAMP-triggered plant immunity. *Current Biology* **28**: 1882–1895.

Tang C, Deng L, Chang D, Chen S, Wang X, Kang Z. 2015. TaADF3, an actin-depolymerizing factor, negatively modulates wheat resistance against *Puccinia striiformis*. *Frontiers in Plant Science* **6**: 1214.

Vilches Barro A, Stockle D, Thellmann M, Ruiz-Duarte P, Bald L, Louveaux M, von Born P, Denninger P, Goh T, Fukaki H *et al.* 2019. Cytoskeleton dynamics are necessary for early events of lateral root initiation in Arabidopsis. *Current Biology* **29**: 2443–2454.

Wang C, Liu Y, Li SS, Han GZ. 2015. Insights into the origin and evolution of the plant hormone signaling machinery. *Plant Physiology* **167**: 872–886.

Yi H, Richards EJ. 2008. Phenotypic instability of Arabidopsis alleles affecting a disease Resistance gene cluster. *BMC Plant Biology* **8**: 36.

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