Leucine-rich repeat extensin proteins regulate plant salt tolerance in *Arabidopsis*

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Contributed by Jian-Kang Zhu, November 2, 2018 (sent for review October 8, 2018; reviewed by Yan Guo and Julio Salinas)

The perception and relay of cell-wall signals are critical for plants to regulate growth and stress responses, but the underlying mechanisms are poorly understood. We found that the cell-wall leucine-rich repeat extensins (LRX) 3/4/5 are critical for plant salt tolerance in *Arabidopsis*. The LRXs physically associate with the RAPID ALKALINIZATION FACTOR (RALF) peptides RALF22/23, which in turn interact with the plasma membrane-localized receptor-like protein kinase FERONIA (FER). The *lrx3*/*lrx4*/*lrx5* triple mutant as well as *fer* mutant plants display retarded growth and salt hypersensitivity, which are mimicked by overexpression of RALF22/23. Salt stress promotes S1P protease-dependent release of mature RALF22 peptides. Treatment of roots with mature RALF22/23 peptides or salt stress causes the internalization of FER. Our results suggest that the LRXs, RALFs, and FER function as a module to transduce cell-wall signals to regulate plant growth and salt stress tolerance.

**Significance**

Plants have evolved cell-wall integrity signaling pathways to maintain cell-wall homeostasis in response to stress conditions, but the components involved in the perception and transduction of cell-wall signals are largely unknown. Here we found that the *Arabidopsis* cell-wall-localized leucine-rich repeat extensins (LRX) 3/4/5 interact with RAPID ALKALINIZATION FACTOR (RALF) peptides RALF22/23. Mature RALF22/23 peptides interact with the plasma membrane-localized FERONIA (FER) and induce FER internalization. The *lrx3*/*lrx4*/*lrx5* triple mutant and *fer* mutant plants overexpressing RALF22 or RALF23, displayed similar phenotypes, including retarded growth and increased sensitivity to salt stress. Salt stress causes the S1P protease-dependent release of mature RALF22 peptides, which in turn induce the internalization of FER via an endosomal pathway. Taken together, our results suggest that the LRXs, RALFs, and FER form a signaling module that connects salt-stress-induced processes, including the preservati

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**Author contributions:** C.Z., O.Z., and J.-K.Z. designed research; C.Z., O.Z., Z.Y., W.J., P.Z., C.-H., and L.Z. performed research; C.Z., O.Z., P.Z., C.-H., and W.A.T. analyzed data; and C.Z., R.L.-D., and J.-K.Z. wrote the paper.

**Reviewers:** Y.G., College of Biological Sciences; and J.S., Consejo Superior de Investigaciones Científicas.

The authors declare no conflict of interest.

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This article contains supporting information online at www.pnas.org/lookup/suppl/doi:10.1073/pnas.1816991115/-/DCSupplemental.

Published online December 4, 2018.
cell-wall changes to the regulation of growth and salt stress
tolerance.

Results

**LRX3, LRX4, and LRX5 Proteins Are Required for Salt Tolerance.** It has
been shown that the Arabidopsis *lrx3 lrx4* (*lrx34*) double and *lrx3 lrx4 lrx5* (*lrx345*) triple mutants are defective in cell-wall composition and display growth retardation (23) (Fig. 1A and B). We found that *lrx3* and *lrx345* mutant plants also displayed increased anthocyanin accumulation, especially in petioles (Fig. 1C and D). To test whether the altered cell wall in *lrx* mutants may affect salt stress tolerance, we grew these mutants in the presence of NaCl and found that both *lrx3* and *lrx345*, but not any of the *lrx* single mutants, exhibited severe salt hypersensitivity (Fig. 1E–G and SI Appendix, Fig. S1A). This salt hypersensitivity phenotype was most dramatic in the *lrx345* triple mutant, which showed significant lethality under 100 mM or more NaCl (Fig. 1F and G). The results suggest that the three LRX proteins function redundantly to promote plant survival under salt stress. The retarded growth and salt-hypersensitive phenotypes of the *lrx345* mutant were complemented by expressing a WT LRX3-coding sequence (CDS) (SI Appendix, Fig. S1B and C), supporting that the *lrx* mutations are the cause of the observed mutant phenotypes.

Because FER is required for cell-wall integrity and salt tolerance (6), we set out to compare the phenotypes of the *fer-4* mutant to those of *lrx345*. We found that the *lrx345* and *fer-4* mutants exhibited very similar phenotypes, including retarded growth, increased anthocyanin accumulation and markedly increased sensitivity to NaCl (Fig. 2). The salt-hypersensitive phenotype of *fer-4* could be complemented by expression of a (WT) FER (SI Appendix, Fig. S2A). These results suggest that LRX3/4/5 and FER may function in the same pathway for growth control and salt tolerance. Of note, *fer-4* mutant plants display additional phenotypes, such as a larger seed size and deficiency in 1-naphthaleneacetic acid–induced root hair development (16, 28), which were not observed in the *lrx345* mutant (SI Appendix, Fig. S2B–D), indicating that some of the functions of FER are not shared by LRX3/4/5.

**The LRX3/4/5 Proteins Interact with RALF Peptides.** To investigate the mechanisms underlying the function of the LRX proteins in growth regulation and salt tolerance, we performed immunoprecipitation–mass spectrometry (IP–MS) analysis using 35S::LRX3–YFP–HA, 35S::LRX4–YFP–HA, and 35S::LRX5–YFP–HA transgenic plants to identify potential interacting partners of the LRXs. The transgenic plants expressing 35S::GFP were used as a control. Interestingly, peptides belonging to the four phylogenetically related RALF peptides RALF22, RALF23, RALF24, and RALF31 (27) were identified in the IP–MS samples from the LRX transgenic plants, but not in the control sample (Fig. 3A and Dataset S1). Split luciferase (split-LUC) complementation assays confirmed the interactions of LRX3 and LRX4 with all these four RALF peptides, and the LRR domain of LRX3 and LRX4 (named LRR3 and LRR4) was sufficient for the interactions (Fig. 3B and SI Appendix, Fig. S3A). We tested whether LRR3 may be able to form a homo-dimer by using the split-LUC assay, but no luciferase activity was detected (SI Appendix, Fig. S3A). Coimmunoprecipitation (Co-IP) assays further confirmed the interactions of LRR3 and LRR4 with RALF22 and RALF23 (Fig. 3C and SI Appendix, Fig. S3B and C), and an in vitro pull-down assay supported that LRR3 directly interacts with RALF22 (SI Appendix, Fig. S3D). In agreement with published results, we found that FER interacts with RALF23 (15), as well as with RALF22 (Fig. 3D). To investigate
whether the FER protein might be physically associated with the LRX proteins, we examined the IP–MS data generated from 35S::LRX3-YFP-HA, 35S::LRX4-YFP-HA, and 35S::LRX5-YFP-HA transgenic plants, but no FER peptides were detected (Dataset S1). We also performed Co-IP assays by expressing LRR3 and ectoFER in Nicotiana benthamiana leaves, but did not observe an interaction between these two proteins in the assay. These results suggest that LRX3/4/5 and FER do not exist in a complex.

RALF22 Overexpressing Plants Phenocopy the lrx345 and fer-4 Mutants.

To understand the biological roles of the RALF peptides associated with LRX3/4/5 and FER, we generated transgenic plants overexpressing the RALF22 gene. Two independent transgenic lines with high expression levels of the RALF22 gene were examined (Fig. 4A). Similar to lrx345 and fer-4 mutants, the transgenic plants overexpressing RALF22 displayed retarded growth, increased accumulation of anthocyanin, and hypersensitivity to NaCl (Fig. 4 B–D). Similar phenotypes were also observed in transgenic plants overexpressing the RALF23 gene (SI Appendix, Fig. S4). We crossed the lrx345 triple mutant with a knockdown mutant of RALF22 (SI Appendix, Fig. S5 A and B) and obtained a quadruple mutant. Our results show that, although the ral22 single mutant did not have an obvious phenotype on salt medium (SI Appendix, Fig. S5 C), the ral22 mutation partially suppressed the salt hypersensitivity of the lrx345 mutant (SI Appendix, Fig. S5 D and E), suggesting that the salt-hypersensitive phenotype of the lrx345 mutant is mediated by the RALF peptides.

Fig. 2. lrx345 and fer-4 mutants show similar phenotypes. (A) Rosette morphology of WT and lrx345 and fer-4 mutants grown in soil for 4 wk. (Scale bar, 1 cm.) (B) Quantification of anthocyanin accumulation in 12-d-old seedlings of WT, lrx345, and fer-4 mutants. Values are means ± SD (n = 3); **P < 0.01 (Student’s t test). (C) Phenotypes of WT, lrx345, and fer-4 seedlings grown on MS and MS + NaCl (120 mM) media. (D) Survival rates of WT, lrx345, and fer-4 seedlings on MS + NaCl (120 mM) medium. Values are means ± SD (n = 3); **P < 0.01 (Student’s t test).

Fig. 3. RALF peptides are associated with LRX3/4/5 and FER. (A) Peptides of LRX and RALF proteins identified in IP–MS assays. Proteins were extracted from 35S::LRX3-YFP-HA, 35S::LRX4-YFP-HA, and 35S::LRX5-YFP-HA transgenic plants. The transgenic plants overexpressing free GFP were used as a control. The isolated proteins were incubated with anti-GFP antibodies overnight and then with protein G for an additional 2 h. The immunoprecipitated samples were analyzed by mass spectrometry. The number of peptides identified for each protein is shown, and a dash “-” indicates that the peptides were not identified. (B) Split luciferase complementation assays showing the interaction between RALF22 and LRX3 or the LRR domain of LRX3 (named LRR3). The constructs to express the indicated fusion proteins were transformed to N. benthamiana leaves through Agrobacterium infiltration. Luciferase activity was determined at 48 h after infiltration. (C) Coimmunoprecipitation assay showing the interaction between RALF22 and LRX3. (D) Coimmunoprecipitation between RALF22 and LRX3 or the LRR domain of LRX3 (named LRR3). The coimmunoprecipitated samples were analyzed by mass spectrometry. The number of peptides identified for each protein is shown, and a dash “-” indicates that the peptides were not identified. (E) IP by anti-HA antibody. Immunoblotting was performed using anti-GFP antibodies.
To test whether salt stress may affect the association between the LRXs and RALFs, we coinfiltrated Agrobacteria expressing LRX3-Nluc and RALF22-cLuc into two spots of one N. benthamiana leaf and sprayed water or 200 mM NaCl on the infiltrated spots. After the treatment for 1 h, the luciferase activities of the infiltrated spots were examined. The spot sprayed with NaCl showed a substantially lower luciferase activity than that sprayed with water (SI Appendix, Fig. S3E), which suggests that the LRXs and RALFs dissociate under salt stress.

RALFs are small secreted peptides that need to be processed to mature active forms via cleavage at sites with the RRXL motif (29), and it has been shown that SITE-1 PROTEASE (S1P) is required for the cleavage (15, 30). Here, we found that salt stress caused an enhanced accumulation of mature RALF22 peptides (Fig. 5A) in a manner that depends on S1P (Fig. 5B). The salt-hypersensitive phenotype of lrx345 mutant seedlings was largely suppressed by slp mutation (Fig. 5C and D), suggesting that the enhanced salt sensitivity of lrx345 mutant plants is mediated by the mature active form of RALF peptides.

**RALF Peptides and Salt Stress Promote the Internalization of FER Protein.** A previous study has shown that RALF23 negatively regulates FER in plant immunity (15). Our results above showed that RALF22 and RALF23 expressing plants phenocopy the loss of function of FER in terms of plant growth and salt sensitivity, suggesting that these RALF peptides may negatively regulate FER function in salt tolerance. Upon ligand binding, several RLKs have been shown to undergo internalization through endocytosis (31–34), which may remove signaling-competent receptor molecules from the membrane pool (35).

To investigate whether the RALF22 and RALF23 peptides may induce the internalization of FER, we treated pFER:FER-GFP transgenic plants with synthesized mature RALF22 and RALF23 peptides and then examined the subcellular localization of FER-GFP in the treated roots. Upon treatment with mature RALF22 and RALF23, FER-GFP could be observed in intracellular compartments in a time-dependent manner, whereas under basal conditions FER-GFP was localized mainly at the plasma membrane (Fig. 6A and SI Appendix, Fig. S6). This observation indicates that mature RALF22/23 can induce the internalization of FER. The internalized FER-GFP was colocalized with the endocytic tracer FM4-64 (Fig. 6B), suggesting that FER proteins traffic along the endosomal pathway. As a control, the bacterial pathogen-associated molecular pattern flagellin (flg22), which can induce the internalization of its cognate RLK FLAGELLIN SENSING2 (FLS2) (31), did not induce the internalization of FER (Fig. 6A). These results suggest that the internalization of FER is induced specifically by the RALF peptides. The internalization of FER-GFP was also detected following treatment with NaCl (Fig. 6C), which is consistent with the NaCl-induced accumulation of mature RALFs.

**Discussion**

Extensive studies in the past few years have demonstrated critical roles of FER in the regulation of multiple cellular processes.
PNAS | December 18, 2018 | vol. 115 | no. 51 | 13127

repair signaling by triggering a rapid cell-autonomous increase in cytosolic [Ca\(^{2+}\)]. The extracellular domain of FER protein can interact with pectin, so FER may directly sense changes in the pectin network (6). LRX proteins consist of an N-terminal LRR domain that binds to RALF peptides and a C-terminal extensin domain (17, 36). Extensins are structural cell-wall proteins that interact with other cell-wall components including pectins through ionic interactions as well as covalent cross-linking (37). The combination of FER and extensin domains places the LRX proteins in an ideal position to sense cell-wall signals and to relay this information to downstream components. The severe salt hypersensitivity of the lrx3 mutants suggests that the LRX3/4/5 are important sensors of cell-wall integrity signals. Under salt stress, cell-wall perturbations may somehow trigger the release of mature RALF peptides, which negatively regulate FER by causing FER internalization.

FER has been identified as the receptor of RALF peptides (7, 15), but how RALF peptides regulate the function of FER is not fully understood. Previous studies have shown that RALF1 activates FER by inducing its phosphorylation (7, 14), but our data indicate that RALF22 and RALF23 peptides may negatively regulate the function of FER in salt tolerance by inducing its internalization. The RALF peptide-induced internalization of FER may be a widespread phenomenon, which might underlie the observation that treatment with mature RALFs can inhibit the scaffolding role of FER in FLS2/EF-BAK1 complexes (15), as well as the inhibitory role of RALF peptides on root elongation and pollen tube growth (7, 27, 38). Ligand-induced activation and internalization have been reported for many other receptor-like kinases (31, 32, 39, 40). One of the well-studied examples in Arabidopsis is FLS2. FLS2 is a pattern recognition receptor, which specifically recognizes a 22-amino-acid epitope of bacterial flagellin (fls22) (41). fls22 induces the association of FLS2 with BAK1 to trigger downstream defense responses (42). Upon activation, FLS2 is internalized via clathrin-dependent endocytosis (31, 32) and finally degraded through an ubiquitination-mediated process (43). Whether the internalized FER is degraded and which E3 ligase may be involved in this process need to be investigated in the future.

In summary, our study suggests that specific cell-wall structural proteins (LRXs), small peptides (RALFs), and a plasma-membrane-localized receptor-like kinase (FER) function as a module to coordinate cell-wall integrity, plant growth, and stress responses by controlling the pool of active FER.

Recently, Feng et al. (6) found that FER protein may sense salt-induced disruption of pectin cross-linking and induce cell-wall

Fig. 6. Mature RALF22 peptide and salt stress promote the internalization of FER protein. (A) Effect of mature RALF22 (mRALF22) treatment on FER-GFP subcellular localization. The pFER::FER-GFP transgenic plants were treated with synthesized mRALF22 (1 μM) or flg22 (1 μM) peptide. Fluorescence in root cells was detected at the indicated time point after treatment using confocal microscopy. (B) pFER::FER-GFP transgenic plants were treated with synthesized mature RALF22 peptide (1 μM) for 1 h. Staining with the endocytic tracer FM4-64 is shown. (C) pFER::FER-GFP transgenic plants were treated with NaCl (150 mM). Fluorescence in root cells was detected at the indicated time point after treatment using confocal microscopy. Identical parameters, including the same laser strength and the same pinhole, were applied for all samples. Arrowheads indicate the internalized compartments. (Scale bars, 10 μm.)

(6, 7, 10, 11, 14, 15). Thus, how the FER protein is able to achieve so many distinct functions is of great interest. In this study, we found that lrx3/4 mutants and fer-4 mutants share some similar phenotypes including retarded growth and increased sensitivity to salt stress, suggesting that LRX3/4 and FER likely function in the same pathways for growth control and salt tolerance. Considering that FER is a plasma-membrane-localized protein (11) and that the LRX proteins function in the cell wall (23), it is likely that FER functions downstream of the LRX proteins, and the phenotypes of lrx3/4 mutants are likely caused by the inhibition of FER function. LRX3/4 interacted with several RALF peptides including RALF22 and RALF23, and transgenic plants overexpressing RALF22 or RALF23 phenocopied lrx3/4 and fer-4 mutants in the aforementioned phenotypes. Application of synthesized RALF22 or RALF23 peptide promoted the internalization of FER, probably via endocytosis. Based on these results, we propose that LRX3/4, RALF22/23, and FER function as a module to sense and transmit cell-wall integrity signals and thereby to regulate plant growth and salt stress response.

In summary, our study suggests that specific cell-wall structural proteins (LRXs), small peptides (RALFs), and a plasma-membrane-localized receptor-like kinase (FER) function as a module to coordinate cell-wall integrity, plant growth, and stress responses. Under normal conditions, the LRX3/4/5 proteins interact with the RALF22/23 peptides, perhaps to prevent the association of the RALF22/23 peptides with the FER protein and thus to inhibit the internalization of the FER protein. Under salt stress, the LRX3/4/5 proteins may directly sense salt-induced changes in the cell wall, and RALF peptides are dissociated from the LRXs to transduce the cell-wall signals to the FER protein. In this module, FER acts as an executor to trigger intracellular signaling (SI Appendix, Fig. S7). This model raises several questions. It is unclear what specific salt-stress-induced cell-wall alterations are sensed by the LRX proteins. It is also unclear whether the LRXs may have a role in the S1P-dependent accumulation of mature RALFs under salt stress. The retarded growth phenotypes of lrx3/4 and fer-4 mutants suggest that the LRX-RALF-FER module represents a major cell-wall-integrity-sensing pathway
for plant growth and that the mutants may be kept alive by unknown residual cell-wall-integrity-sensing pathways. It is possible that salt-stress-induced signaling may somehow inhibit the residual cell-wall-integrity-sensing and –repair pathways that remain in the lrx345 and fer mutants, causing complete growth inhibition and plant death.

Materials and Methods

Plant Materials

The Col-0 ecotype of Arabidopsis thaliana was used as the WT. Plants were grown at 23 °C with a long-day light cycle (16 h light/8 h dark). The lrx3 (SALK_094400), lrx5 (GABI_017A08), lrx5 (SALK_013968), lrx34, lrx45, fer-4, and 1sp mutants have been described previously (7, 23, 30). The rlr22 mutant (GK-293909) was obtained from the Arabidopsis Biological Resource Center (Ohio State University). The 1sp lrx345 and rlr22 lrx345 quadruple mutants were generated by crossing. Homozygous mutants were confirmed by PCR-based genotyping. Transgenic plants containing 35S:LRX1- YFP-HA, 35S:LRX5-YFP-HA, 35S:LRX5-YFP-HA, 35S:RALF22-YFP-HA, and 35S:: RALF23-YFP-HA expression cassettes were generated by Agrobacterium tumefaciens-mediated transformation. The primers used for genotyping are listed in SI Appendix, Table S1.

Construction of Plasmids

To generate constructs for transgenic plants and protein interaction assays, the whole corresponding CDSs of the LRX proteins were amplified from cDNA using gene-specific primers (SI Appendix, Table S1), and the amplified fragments were subcloned into pDONR207 ENTRY using BP clone II kit (Life Technologies). The inserted fragments were recombined to destination vectors using LR donase II kit (Life Technologies). The generated constructs were stably transformed to Arabidopsis or transiently transformed to N. benthamiana by Agrobacterium tumefaciens-mediated transformation. For the pET-28a-mRALF22 construct, the frameshift encoding sequence of the peptide of RALF22 was amplified and cloned into the vector pET-28a by using BamHI and Xhol restriction sites. For the pGEX-4T-1-LRR3 construct, the LRR3 fragment was amplified and cloned into the vector pGEX-4T-1 by using SalI and NotI restriction sites.

Antioxidant Measurement

Antioxidant content was assessed as previously described (45). In brief, 30 seedlings for each sample were collected in a 2-μL Eppendorf tube with 600 μL of 1% HCl in methanol (vol/vol). The seedlings were incubated overnight in the dark at 4 °C with gentle shaking. After extraction, 400 μL of water and 400 μL of chloroform were added, and the samples were vortexed. The tubes were centrifuged at 12,000 × g for 2 min, and the supernatant was transferred to a new tube. The absorbance of each supernatant was measured spectrophotometrically at 530 and 657 nm, respectively. The concentration of ascorbic acid was calculated using the formula:

\[ \text{AsA} = 0.25 \times \text{Abs}2 \]

Recombinant protein expression, in vitro pull-down, split luciferase complementation assay, synthetic peptides, RALF22 cleavage assay, coimmunoprecipitation assay, fluorescence assay, and LC-MS/MS analysis are described in SI Appendix, SI Materials and Methods. The sequences of mature RALF22 and RALF23 are listed in SI Appendix, Table S2.

Acknowledgments

We thank Rebecca Stevenson for technical assistance, Prof. Christoph Ringli for providing lrx34 and lrx345 seeds, Prof. Alice Y. Cheung for providing pFER:FER-GFP seeds, and Prof. Yuwei Jiang for assistance with anthocyanin measurement. This work was supported by the Strategic Priority Research Program (Grant XDB27040000) of the Chinese Academy of Sciences.

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