Supporting Information

Liu et al. 10.1073/pnas.1215543110

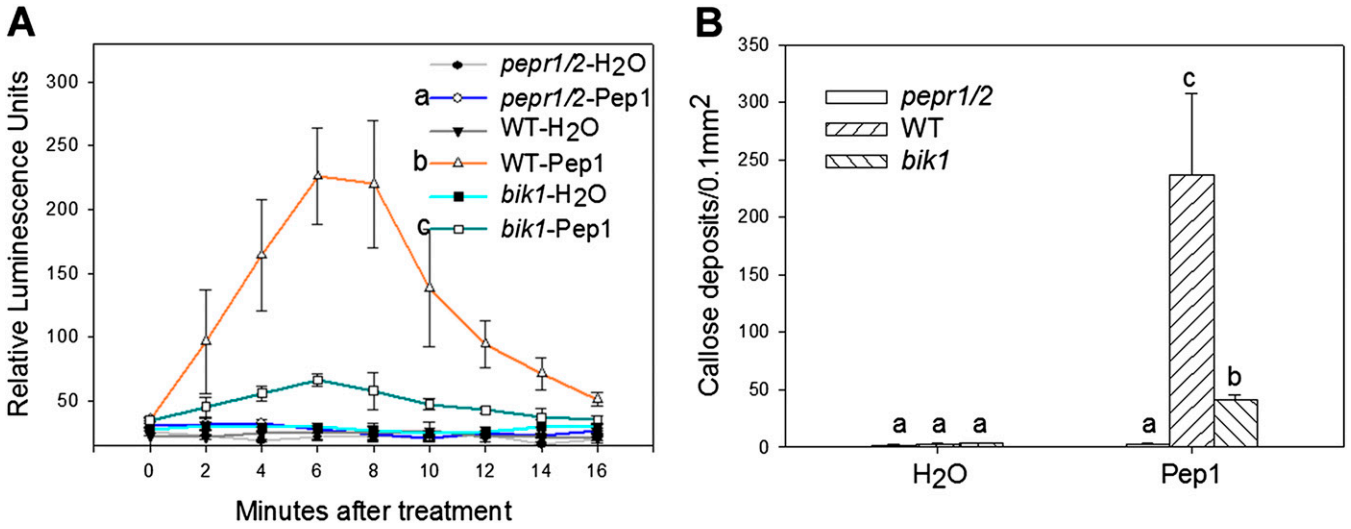

Fig. S1. *Botrytis-induced kinase 1 (bik1)* is compromised in Pep1-induecd defenses. (*A*) H_2O_2 production in leaf strips of *bik1*, *pep1 receptor kinase 1/pep1 receptor kinase 2* (*pepr1/2*), and WT treated with 1 μM Pep1. Different letters indicate significant difference (mean ± SD; $n \geq 4$; $P < 0.01$). (*B*) Callose deposition in *bik1*, *pepr1/pepr2* (*pepr1/2*), and WT leaves treated with 1.5 μM Pep1. Different letters indicate significant difference (mean + SD; $n \geq 16$; $P < 0.01$).

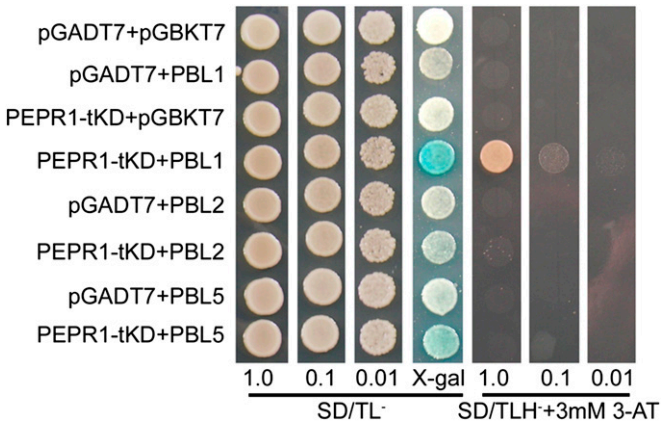

Fig. S2. Interaction of PBS1-like proteins (PBLs) with PEPR1. The truncated PEPR1 KD (PEPR1-tKD) prey plasmid was cotransfected into yeast with different PBL bait plasmids and tested for *lacZ* and *His* reporter activity as described Fig. 1*A*.

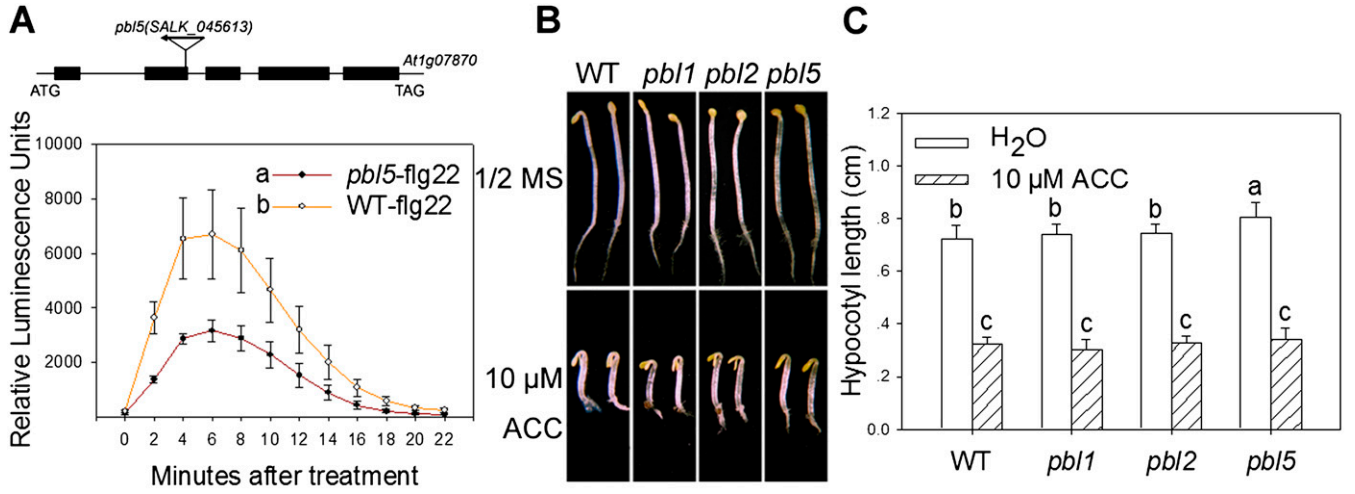

Fig. S3. (*A*) The *pbl5* mutant is compromised in flg22-induced H_2O_2 accumulation. (*Upper*) Schematic representation of T-DNA insertion in the *PBL5* gene. Black boxes denote annotated exons from TAIR. The triangle and arrowhead indicate position and direction of T-DNA insertion, respectively. (*Lower*) *pbl5* plants are compromised in flg22-induced oxidative burst. Different letters denote significant difference (mean ± SD; $n \geq 4$; $P < 0.01$). (*B* and *C*) *pbl1*, *pbl2*, and *pbl5* mutants display normal triple response. (*B*) Photograph of 5-d-old WT, *pbl1*, *pbl2*, and *pbl5* etiolated seedlings germinated on 1/2 MS medium with or without 10 μM 1-aminocyclopropane-1-carboxylate (ACC). (*C*) Hypocotyl length of the seedlings (mean + SD; $n \geq 12$; $P < 0.01$).

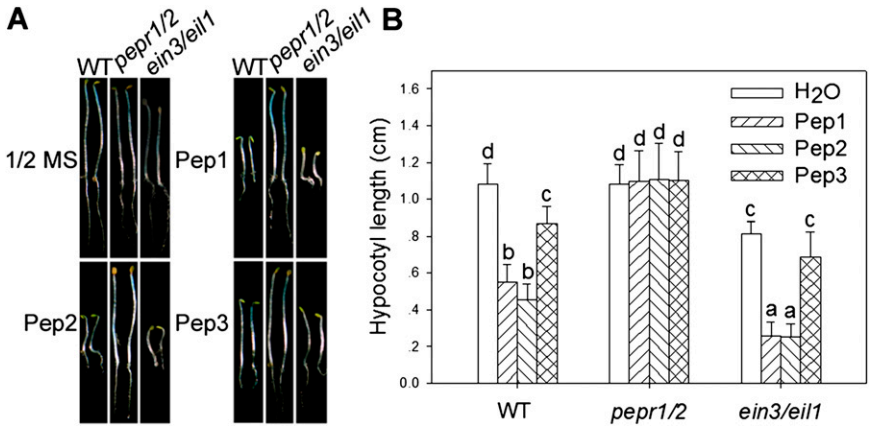

Fig. S4. Pep1-3 inhibits hypocotyl and root growth. (*A*) Photograph of 5-d-old etiolated seedlings grown in the presence of 10 μM Pep1, Pep2, and Pep3. (*B*) Hypocotyl length. Different letters denote significant difference (mean + SD; $n \geq 17$; $P < 0.01$).

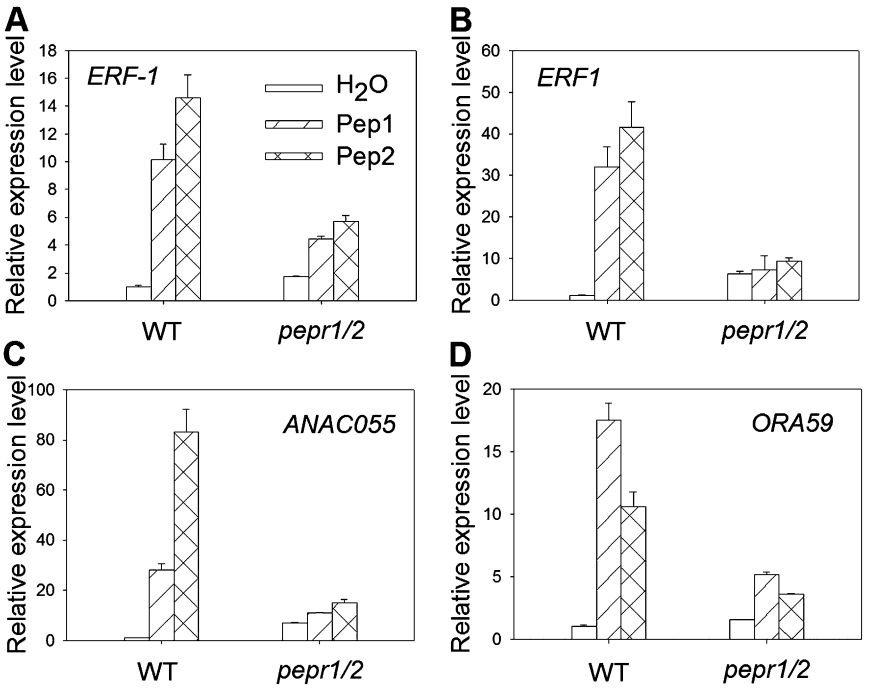

Fig. S5. Pep1 and Pep2 induce defense-related genes expression. (*A–D*) Real-time RT-PCR analysis of Pep1- and Pep2-induced *ERF-1*, *ERF1*, *ANAC055*, and *ORA59* genes expression. The 8-d-old WT and *pepr1/pepr2* seedlings were sprayed with H_2O or 10 μM Pep1 or Pep2 containing 0.01% silwet L-77, and RNA was isolated 10 h later.

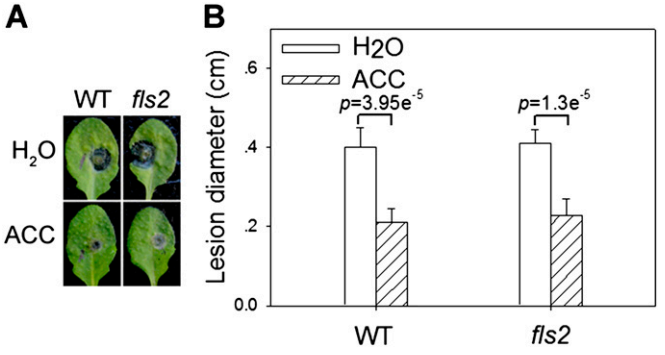

Fig. S6. FLS2 is not required for ACC-induced resistance to *Botrytis cinerea*. (*A*) Symptoms 2 d after *B. cinerea* inoculation. The experiments were performed as described in Fig. 5*E*. (*B*) Lesion size of indicated plants (mean + SD; $n \geq 7$). *P* value is given above the bars.

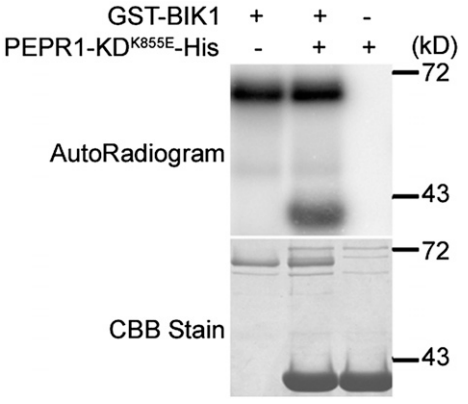

Fig. S7. BIK1 phosphorylates PEPR1 KD in vitro. His-tagged PEPR1 KD^{K855E} was incubated with GST-tagged BIK1 in kinase buffer containing ^{32}P-γ-ATP, and protein phosphorylation was detected by autoradiography. CBB-stain shows protein loading. The experiment was performed as described in Fig. 6*A*.

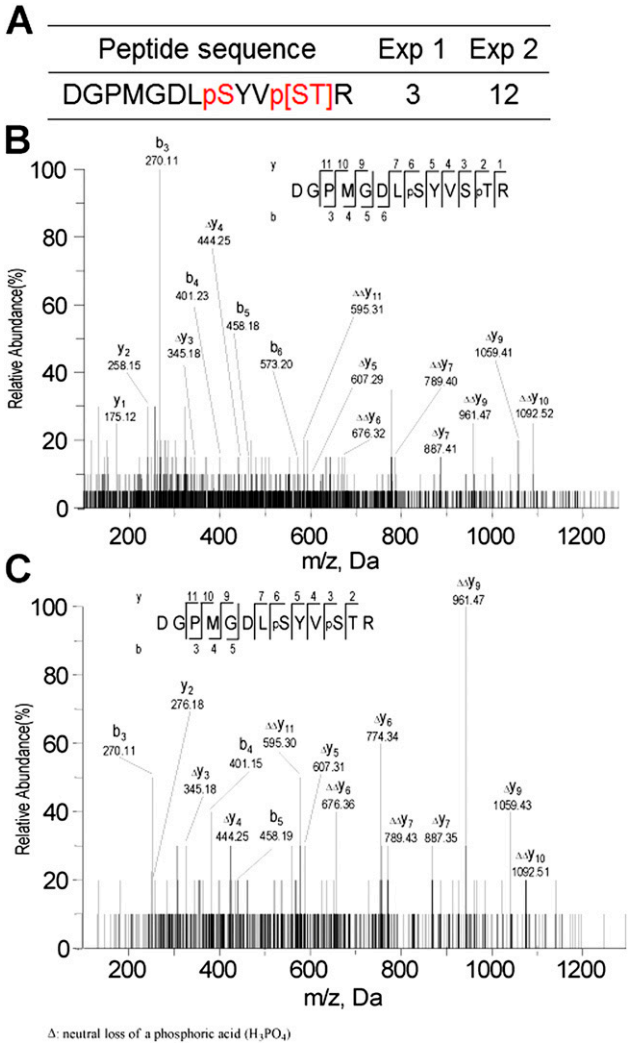

Fig. S8. Identification of BIK1 amino acids phosphorylated by PEPR1 KD. The GST-BIK1^{K105E} protein, coexpressed with PEPR1-KD-His in *Escherichia coli*, was isolated and subjected to LC-MS/MS analysis. (*A*) Number of phosphorylated peptides corresponding to the activation loop identified in two independent experiments. Parenthesis indicates that the phosphorylation on serine 236 or threonine 237 cannot be easily distinguished. (*B* and *C*) Representative collision-induced dissociation (CID) spectra of the phosphopeptide DGPMGDLpSYVp[ST]R. The major b- and y-type ions are indicated in the mass graphs. Δ, neutral loss of a phosphoric acid.

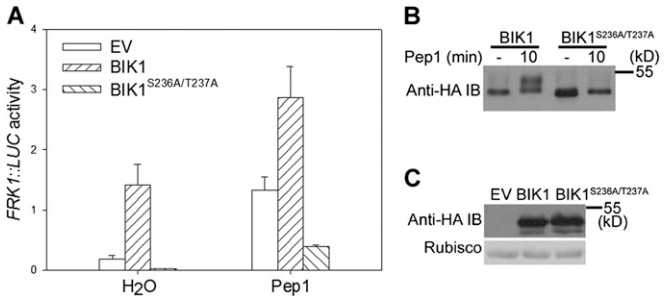

Fig. S9. Serine 236 and threonine 237 are required for Pep1-induced BIK1 phosphorylation and signaling. (*A*) Ala substitution of Ser-236 and Thr-237 in BIK1 blocks Pep1-induced *FRK1* reporter expression in protoplasts. Empty vector (EV), WT BIK1, and BIK1$^{S236A/T237A}$ plasmids were transfected into WT protoplasts along with *35S::R-LUC* and *FRK1::LUC*, treated with H_2O or 1 μM Pep1 for 3 h, and the *FRK1::LUC* activity was determined. (*B*) The BIK1$^{S236A/T237A}$ mutant is not phosphorylated upon Pep1 treatment. (*C*) BIK1 and BIK1$^{S236A/T237A}$ protein accumulation in *A*.