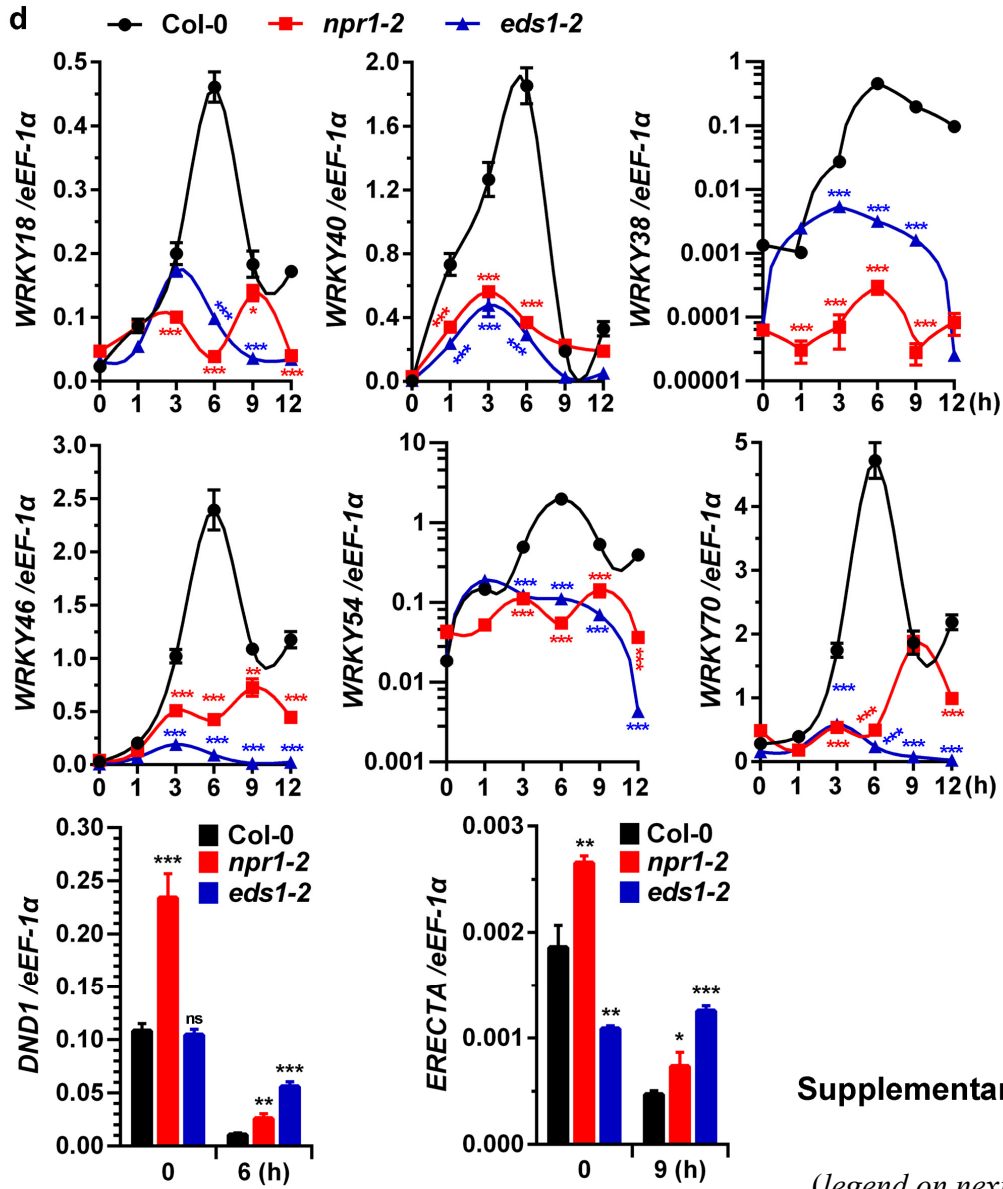
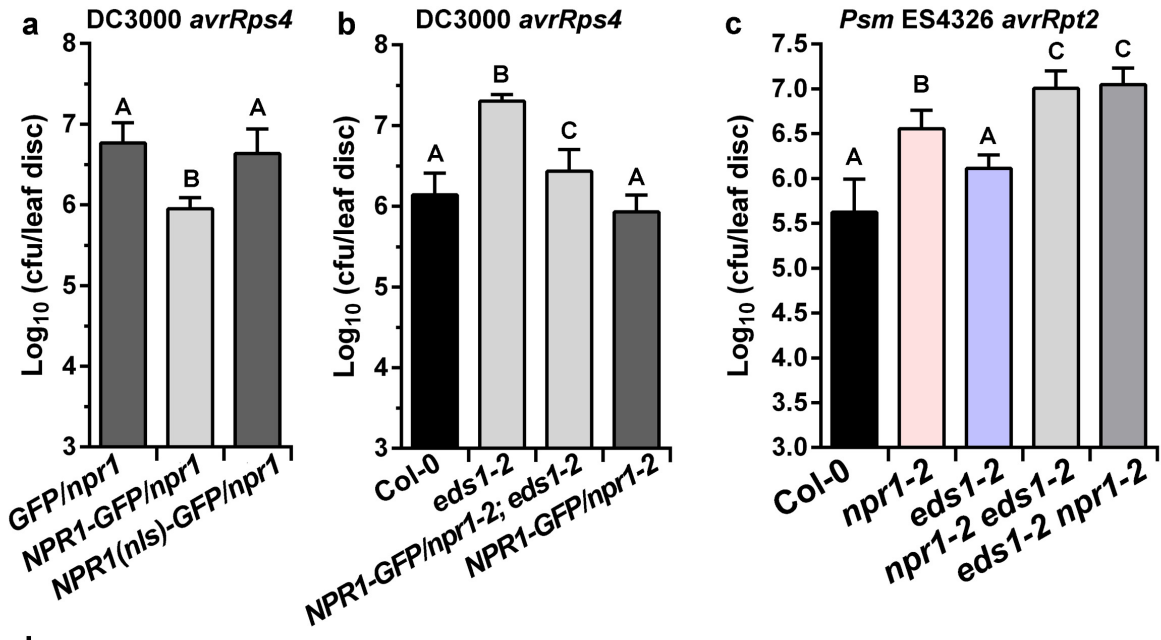


Supplementary Fig. 1

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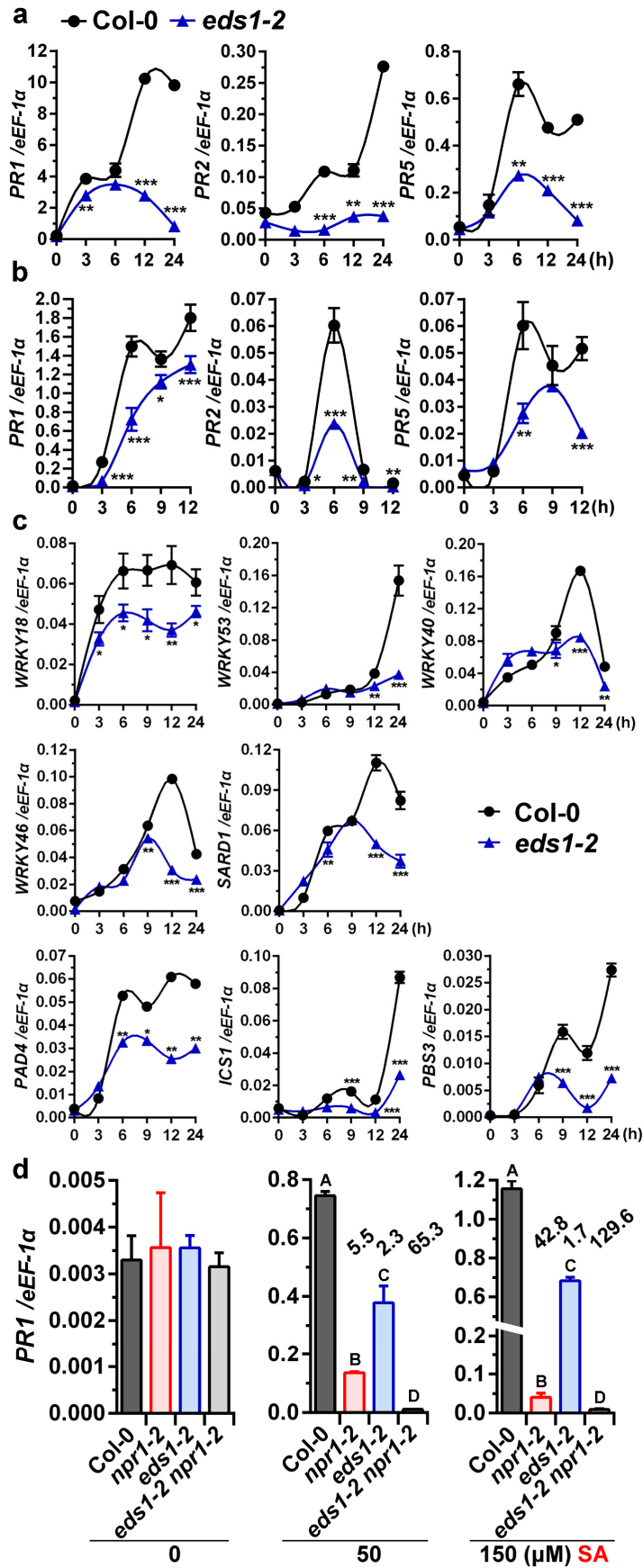
Supplementary Fig. 1 | Identification of the interaction domains in NPR1 and EDS1. **a**, NPR1 specifically interacts with EDS1. The yeast strain Y187 with full-length NPR1 fused with GAL4-DBD was mated with yeast strain AH109 harboring GAL4-AD fusion protein (GUS, PAD4 or SAG101), and then the diploid yeast cells were grown on synthetic double dropout agar media (DDO, without Leu and Trp) and selective quadruple dropout medium (QDO, without Leu, Trp, His, and Ade). GAL4-DBD, GAL4-DNA binding domain; AD, activation domain. **b**, NPR1 recognizes the α -helix region (310-330) in EDS1. Left panel is schematic diagram of the EDS1 deletion (Del) fragments. The numbers indicate the corresponding amino acid residues. The specific regions in EDS1, such as a lipase-like domain, a region (480-540) partially overlapping with EP (EDS1 and PAD4-defined) domain, a coiled-coil domain (358-383) defined by Pfam and MARCOIL, a region (310-350) encompassing two α -helices defined by Phyre², the region (1-273) that is almost equivalent to the left-over region in the null mutant *eds1-2*, a fast neutron-derived mutant containing truncated product S276 (serine 276)-Stop²⁵, and others, are shown in the diagram. Yeast cells were grown on DDO and triple dropout medium (TDO, without Leu, Trp and His) plus 1 mM 3-aminotriazole (3-AT). Diagram is drawn to scale. **c**, Overview of EDS1 protein 3D structure (PDB: 4NFU). The protein ribbon model with diverse structures is illustrated by Accelrys Discovery Studio Visualizer. The protruding interaction α helices JK (310-330) for NPR1-EDS1 interaction is highlighted in yellow. Other structures such as α G, α H, α L and α M are marked in white. Detail information of α helices are described as previously²⁹. **d,e**, EDS1 dynamically interacts with NPR1. The left panel shown is a schematic diagram of the NPR1 deletion (Del) mutants. The numbers indicate the corresponding amino acid residues. BTB/POZ, Broad complex, Tramtrack, and Bric-a-brac/Pox virus and Zinc finger domain; ANK, ankyrin repeats; CTD, a C-terminal domain; TA, transactivation domain; C521A/C529A, the Ala substitution of two residues (Cys 521 and Cys 529) that are critical for TA; NLS, nuclear localization signal. Note that the N-terminal BTB/POZ domain and a second repression region (463-513) in CTD confer the transcriptional repression activity of NPR1 upon SA induction and under normal conditions, respectively²². Diagram is drawn to scale. Modelled structure of NPR1 was generated by Phyre² server using the template c4cj9A (**e**). PyMol was used to visualize and arrange modelled structure.



Supplementary Fig. 2

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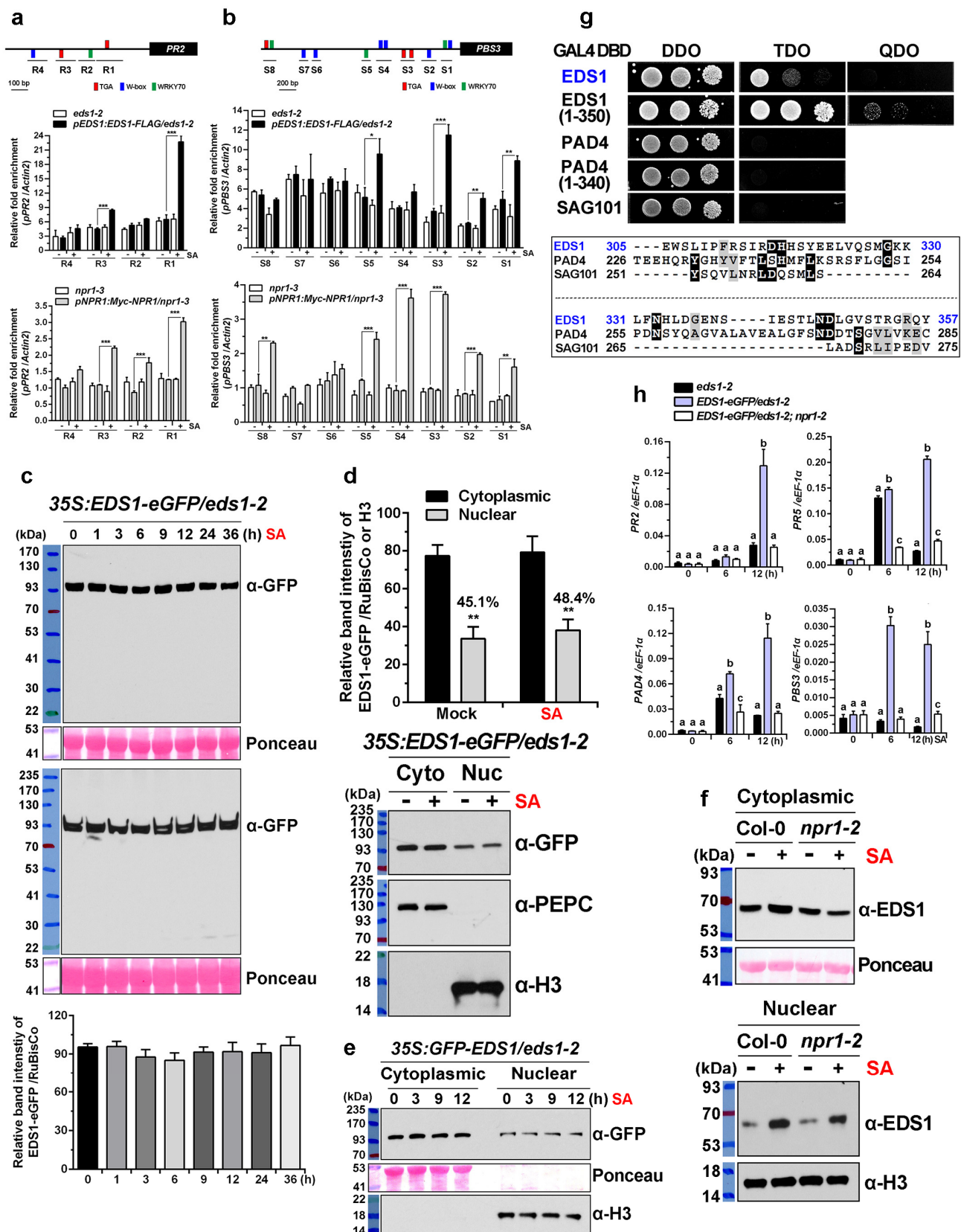
Supplementary Fig. 2 | Genetic and molecular interactions between NPR1 and EDS1 in ETI. **a**, Nuclear NPR1 is involved in ETI. Bacterial growth assays were performed using transgenic *Arabidopsis* constitutively expressing *GFP*, *NPR1-GFP* or *NPR1 (nls)-GFP* under the control of *CaMV* 35S promoter in the *npr1-2* background. **b**, NPR1 functions as a partner of EDS1 in TIR-NB-LRR R protein (RRS1S/RPS4)-activated ETI. Growth of bacterial was determined in Col-0, *eds1-2*, *35S:NPR1/npr1-2;eds1-2* and *35S:NPR1-GFP/npr1-2* transgenic plants. **c**, EDS1 functions as a partner of NPR1 in CC-NLR protein (RPS2)-activated ETI. Leaves of soil-grown plant were inoculated with *Pst* DC3000 *avrRps4* (OD₆₀₀ = 0.0005) in **(a)** and **(b)** or *Psm* ES4326 *avrRpt2* (OD₆₀₀ = 0.0002) in **(c)**; the bacterial titers were measured at 2 dpi (CFU, colony-forming units). Error bars represent standard deviation (SD); n = 6 biologically independent samples **(a-c)**. Statistically significant difference is indicated by different lowercase letters ($P < 0.01$). **d**, NPR1 and EDS1 co-regulate a set of common target genes in RRS1S/RPS4-activated ETI. EDS1-induced *WRKY* genes (*WRKY18*, *WRKY40*, *WRKY38*, *WRKY46*, *WRKY54* and *WRKY70*) and EDS1-repressed genes (*DND1* and *ERECTA*) were analyzed using real-time qPCR. Leaves from 4-week-old soil-grown plants were inoculated with the avirulent bacterial pathogen *Pst* DC3000 *avrRps4* (OD₆₀₀ = 0.01). Total RNA was extracted from inoculated leaves collected at the indicated time points. Expression of *WRKY38* and *WRKY54* was plotted on a log₁₀ scale; gene expression levels were normalized against the constitutively expressed *eEF-1α*. Error bars indicate the (±) SD (n = 3 biologically independent samples). Statistical difference is shown between Col-0 and single mutant (*npr1-2* or *eds1-2*) plants (*t*-test, *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$). ns, no significant difference.



Supplementary Fig. 3

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Supplementary Fig. 3 | EDS1 and NPR1 coregulate some SA-responsive genes. **a,b**, EDS1 can function as a positive regulator of SA signaling (downstream of SA). Total RNA was extracted from rosette leaves of 4-week-old soil-grown plants (**a**) infiltrated with 0.3 mM SA or from 2-week-old seedlings grown on one-half strength Murashige and Skoog (1/2 MS) solid agar media (**b**) exogenously treated with hydroponic 0.5 mM SA solution. Expression of *PR1*, *PR2*, and *PR5* in Col-0 and *eds1-2* was analyzed using real-time qPCR. **c**, EDS1 upregulates some NPR1-mediated and SA-responsive genes. Total RNA was extracted from 2-week-old seedlings on 1/2 MS media exogenously treated with hydroponic 0.5 mM SA solution. Expression of a subset of *WRKY* genes (*WRKY18*, *WRKY40*, *WRKY46*, and *WRKY53*), SA accumulation (*SARD1* and *PAD4*) and biosynthesis (*ICS1* and *PBS3*) genes were analyzed using qPCR. Expression was normalized against constitutively expressed *eEF-1a*. Error bars represent \pm SD; n = 3 biologically independent samples (**a-c**). Statistical significance comparisons between Col-0 and *eds1-2* for each time point were shown on the bars (*t*-test, *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$). **d**, NPR1 and EDS1 synergistically activate *PR1* in response to SA. Total RNA was isolated from 2-week-old seedlings grown on 1/2 MS agar plates containing different low concentrations of SA (50 and 150 μ M) for long-term treatment (mimic SAR treatment). Error bars represent SD (n = 4 biologically independent samples). Different letters indicate statistical difference (two-way ANOVA, $P < 0.01$). The folds above the error bars indicate the fold change for the gene expression reduction compared with the value obtained in Col-0.

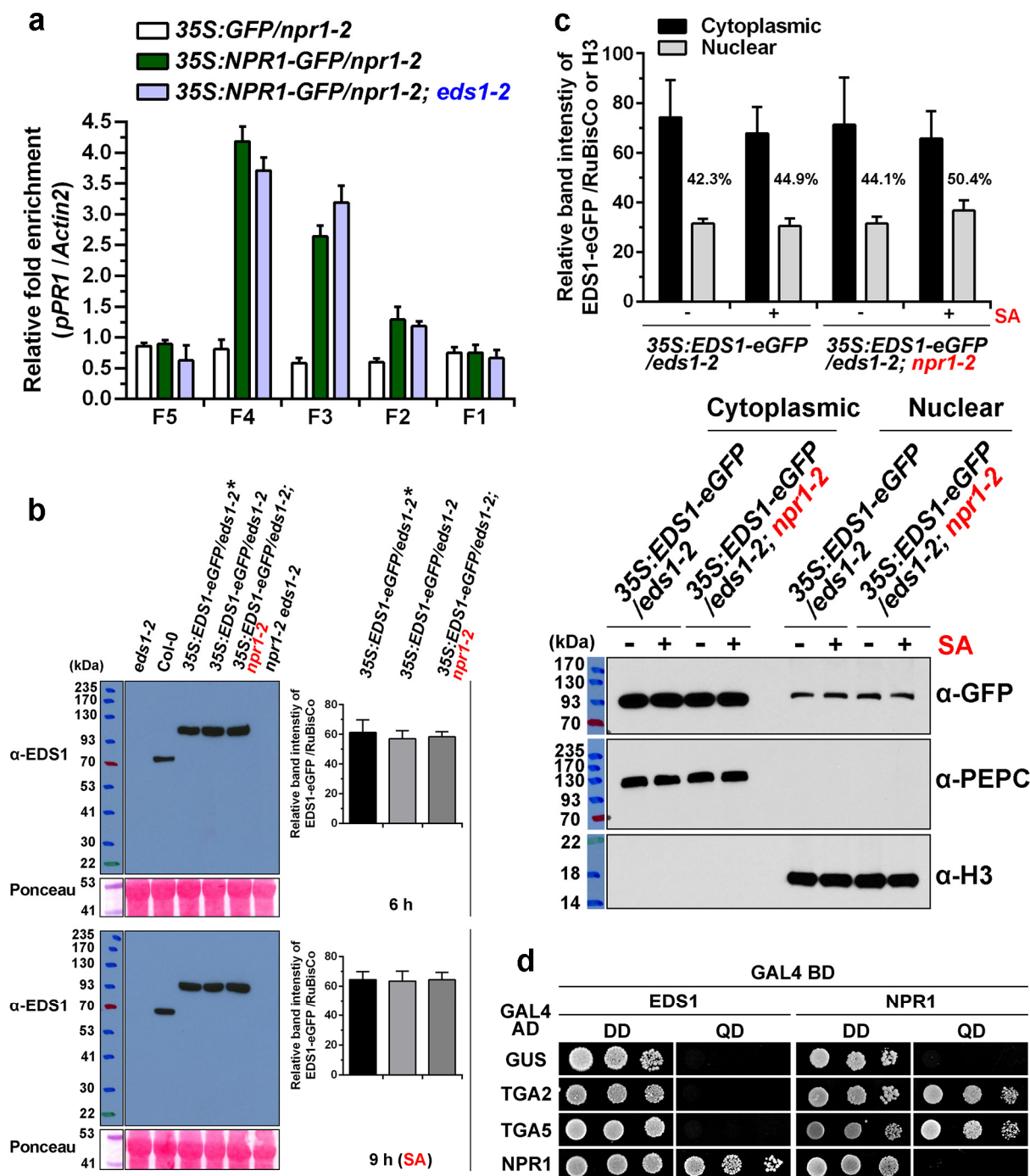


Supplementary Fig. 4

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Supplementary Fig. 4 | Recruitment of NPR1-EDS1 complex onto promoters of plant defense genes upon SA induction. a,b, The NPR1-EDS1 protein complex occupies specific regions of the *PR2* and *PBS3* promoters. ChIP-qPCR shows enrichment of NPR1 and EDS1 at the *PR2* (a) and *PBS3* (b) genomic loci. The mutant control and transgenic plants were harvested after 0.5 mM SA (+) or water (-) treatment for 9 h. After sonication and clearance, ChIP assays were performed with anti-FLAG magnetic beads or anti-Myc antibody. The amount of immunoprecipitated DNA was quantified by qPCR using primers specific to each amplicon. Samples from *npr1-2* or *eds1-2* mutants were used as negative controls. Specific enrichment of fusion protein on genomic DNA sequences was calculated relative to that on *Actin2* coding DNA sequences. Error bars indicate SD (n =3 biologically independent samples). Significances of differences are shown (*t*-test, *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$). Top panel is the schematic representation of the *cis*-elements and chromatin fragments (R1 to R4 for *PR2* and S1 to S8 for *PBS3*) of amplicon in the genomic regions. Diagram is drawn to scale. Details on the positions of the primers are provided in Supplementary Table 1. W-box (blue box), TTGACT/C; TGA motif (red box), TGACG; TGA2/W (chimeric color), TGA2TT (TGA2 binding sites overlapping W-box); WRKY70 (green box), GACTTTT (putative WRKY70 binding sites)⁵². The perfect *cis*-elements with inverted consensus sequences are shown below the promoter. **c,** SA does not induce constitutively expressed EDS1-eGFP. Transgenic plants constitutively expressing EDS1-eGFP under the control of the *CaMV* 35S promoter in the *eds1-2* background (*35S:EDS1-eGFP/eds1-2*) were treated with 0.5 mM SA solution and collected at the indicated time points. Total protein was analyzed in western blotting using anti-GFP antibody. The ribulose-1,5-bisphosphate carboxylase/oxygenase (RuBisCo) large subunit bands visualized by Ponceau S staining are shown as protein loading controls. Lower panel shows relative EDS1-eGFP band intensity of immunoblot analysis. Blots were quantitated using the ImageJ software. Protein level was normalized to the constitutively expressed RuBisCo. Bars indicate SE from three independent blots. **d,e,** SA does not facilitate nuclear translocation of EDS1. The *35S:EDS1-eGFP/eds1-2* transgenic plants (d) and *35S:GFP-EDS1/eds1-2* plants (e) were treated with 0.5 mM SA solution. The former plants were harvested 9 h after treatment for cell fractionation; the latter were collected at the indicated time points. EDS1-eGFP and GFP-EDS1 proteins were immunologically detected using an anti-GFP antibody. Anti-phosphoenolpyruvate carboxylase (PEPC) and Ponceau S staining were used for detection of cytoplasmic (Cyto) markers; anti-Histone H3 antibody was used for detection of the nuclear (Nuc) marker. Upper panel, relative EDS1-eGFP band intensity of immunoblot analysis. Blots were quantitated using the ImageJ software. Cytoplasmic protein level was normalized to the constitutively expressed RuBisCo; nuclear protein level was normalized to histone H3. Bars indicate SE from four independent blots. Significances of differences between cytoplasmic and nuclear fractions are analyzed for each treatment (*t*-test, **, $P < 0.01$). **f,** SA induces the accumulation of endogenous EDS1 in cytoplasm and nuclei. Nucleo-cytoplasmic distribution of endogenous EDS1 protein in SA-treated plant cells was performed using 5-week-old soil-grown plants that were harvested after 12 h treatment with foliar sprays of 0.5 mM SA solution. EDS1 and histone H3 proteins were immunologically detected using anti-EDS1 and anti-histone H3 antibodies, respectively. **g,** EDS1 specifically possesses autoactivation, but not PAD4 or SAG101. The yeast strain Y187 with indicated protein fused with GAL4-DBD was mated with yeast strain AH109 harboring GUS fused with GAL4-AD. Diploid yeast cells were grown on synthetic dropout agar media such as DDO (double dropout; without Leu, Trp), TDO (triple dropout; without Leu, Trp, His) and QDO (quadruple dropout; without Leu, Trp, His, and Ade). Bottom, multiple sequence alignment of NPR1-interacting domain (310~330) and TAD (331~350) of EDS1 among EDS1 and its partners using ClustalW and BoxShade servers (SIB Swiss Institute of Bioinformatics). The numbers indicate the corresponding amino acid residues. **h,** SA-induced

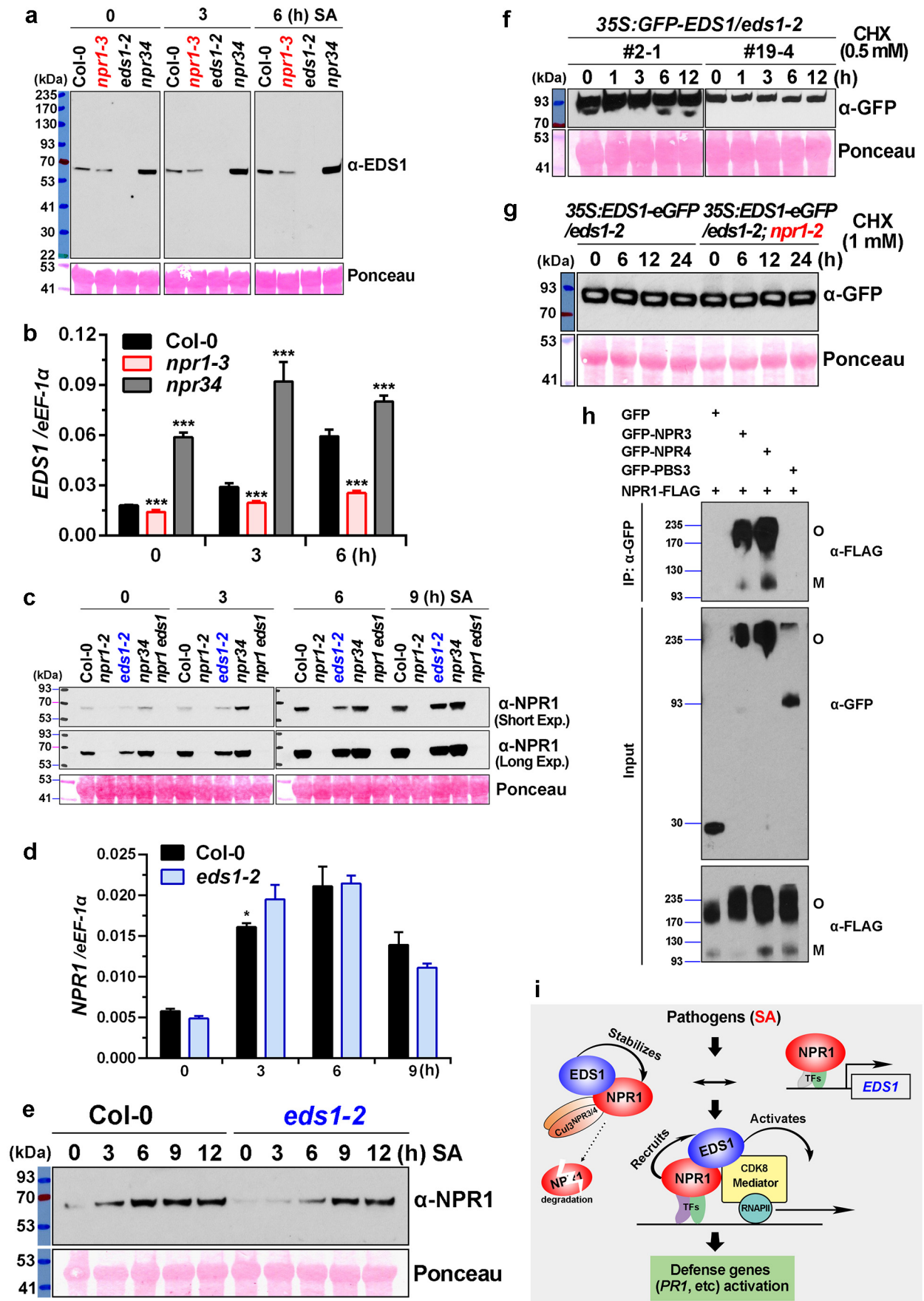
EDS1 activates plant defense genes. Expression of *PR2*, *PR5*, *PAD4* and *PBS3* was analyzed by real-time qPCR. Expression was normalized against constitutively expressed *eEF-1a*. Four-week-old soil-grown plants were infiltrated with 0.5 mM SA solution and leaf tissues were collected at indicated time points. Bars represent SD; n = 4 biologically independent samples. Different letters indicate significant differences (Two-way ANOVA, Tukey's multiple comparisons test, $P < 0.05$). The statistical comparisons were made separately among *eds1-2*, *35S:EDS1-eGFP/eds1-2* and *35S:EDS1-eGFP/eds1-2;npr1-2* for each time point.



Supplementary Fig. 5

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Supplementary Fig. 5 | A physical NPR1-EDS1 interaction confers the recruitment of EDS1 to *PR1* promoter. **a**, EDS1 slightly influences the association of NPR1-GFP with the *PR1* promoter. Four-week-old soil-grown transgenic plants were treated with foliar sprays plus soil drenches of 0.5 mM SA solution for 9 h. The collected leaf tissues were subjected to ChIP assay using GFP-Trap magnetic beads. Schematic representation of the chromatin fragments (F1 to F5) is illustrated in Fig. 4a. Bars indicate SD (n = 3 biological independent samples). This experiment was repeated twice with similar results. **b**, NPR1 has no significant effect on the expression of EDS1-eGFP. Total protein prepared from indicated genotypes was analyzed by immunoblotting using an anti-EDS1 antibody. Relative band intensity was analyzed by normalizing EDS1-eGFP protein against the corresponding RuBisCo. Error bars indicate SD from triplicate technical repeats. The *35S:EDS1-eGFP/eds1-2* denoted by asterisk (*) is the original homozygous transgenic line that was used to cross with *npr1-2*; the resulting homozygous progenies (i.e. *35S:EDS1-eGFP/eds1-2* and *35S:EDS1-eGFP/eds1-2; npr1-2*) from same generation were identified. **c**, NPR1 does not facilitate nuclear translocation of EDS1-eGFP. The indicated transgenic seedlings were treated with 0.5 mM SA (+) or water (-) for 9 h. Relative band intensity and immunoblotting are performed as described in Supplementary Fig. 4d-f. Percentage indicates the ratio of nuclear to cytoplasmic distribution. Bars indicate SE from four independent experiments. **d**, The interaction between EDS1 and TGA2/5 is not detected in Y2H assays. Yeast cells were grown on synthetic double dropout agar media (DD, without Leu and Trp) and selective quadruple dropout medium (QD, without Leu, Trp, His, and Ade). GAL4 BD, GAL4-DNA binding domain; AD, activation domain.



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Supplementary Fig. 6 | NPR1 transcriptionally upregulates *EDS1* expression, while EDS1 stabilizes NPR1 protein. **a,b**, NPR1 transcriptionally upregulates *EDS1* expression. EDS1 protein level (**a**) and EDS1 transcription level (**b**) are analyzed in Col-0, *npr1-3*, *eds1-2* and *npr3-2 npr4-2* (*npr34*) seedlings exogenously treated with hydroponic 0.5 mM SA solution for indicated times. **c-e**, EDS1 stabilizes NPR1 in SA-mediated defense responses. NPR1 protein level (**c**) and *NPR1* transcription level (**d**) are investigated in Col-0, *npr1-2*, *eds1-2*, *npr3-2 npr4-2* (*npr34*), and *npr1-2 eds1-2* (*npr1 eds1*) seedlings treated with 0.5 mM SA solution for indicated times. The short exposure and long exposure (Exp.) of same protein gel blot are shown. Total RNA was extracted from 2-week-old 1/2 MS media-grown seedlings and subjected to real-time qPCR analysis (**b,d**). The *eEF-1 α* gene was used as an internal control. **f,g**, EDS1 protein turnover is not affected by NPR1. The constitutively expressed EDS1 fusion proteins (GFP-EDS1 and EDS1-eGFP) in indicated transgenic seedlings are constant in the presence of cycloheximide (CHX) even at high concentrations (0.5 and 1 mM) and for a long duration (24 h). **h**, Oligomeric and monomeric NPR1 associates with NPR3/4 in plants. *N. benthamiana* was co-transformed with indicated constructs such as *35S:GFP*, *35S:GFP-NPR3/4/PBS3* and *pNPR1:NPR1-3xFLAG*. Approximately 0.2 g of leaf tissues was used to total protein extraction. Co-IP and immunoblotting assays were performed under reducing conditions. O, oligomeric GFP-NPR1/3/4; M, monomeric GFP-NPR1/3/4. **i**, A working model of EDS1-mediated functions in plant immunity. Under plant-pathogen interactions and SA induction, EDS1, acting as a transcriptional coactivator, is directed recruited by SA-induced NPR1 onto promoters of defense genes, and in turn EDS1 cooperates with NPR1 and directly interacts with Mediator complex (e.g. CDK8 module) to facilitate the assemble of RNAPII preinitiation complex, thereby potentiating transcription of downstream defense genes. In addition, EDS1 prevents NPR1 from degradation under normal and immune induced conditions, providing optimal NPR1 level to further immune responses. Simultaneously NPR1 directly targets *EDS1* and upregulates its transcription upon immune induction. Bars indicate (\pm) SD; n = 4 biological independent samples (**b,d**). Statistical significances of difference from control were shown for each time point (*t*-test, *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$). Total protein was analyzed by reducing SDS-PAGE and immunoblotting using indicated proteins. RuBisCo protein stained by Ponceau S was used as internal control. Protein sizes marked on the left are in kDa.

Supplementary Table 1. Primers Used in This Study

Primer Name	Primer Sequence (5' - 3')
Plasmid and Cloning	
GW_F (attB1)	ggggACAAGTTTGTACAAAAAAGCAGGCTtc
GW_R (attB2)	gggcACCACTTTGTACAAGAAAGCTGGGTc
pNPR1:NPR1	GW_F_TTTATGAATATTTAATCTGATTTTTTGGCT GW_R_CCGACGACGATGAGAGAGTTTAC
pEDS1:EDS1	GW_F_GTTTATCAGATTCCACGTACGAT GW_R_GGTATCTGTTATTTTCATCCATCA
EDS1	GW_F_ATGGCGTTTGAAGCTCTTACC GW_R_GGTATCTGTTATTTTCATCCATCA
PAD4	GW_F_ATGGACGATTGTCGATTTCGAG GW_R_AGTCTCCATTGCGTCACTCTC
SAG101	GW_F_ATGGAGTCTTCTTCTTCACTAAAAG GW_R_TTGTGACTTACCATAACTCTCGT
EDS1Δ480-540	EDS1_F_ATGGCGTTTGAAGCTCTTACC EDS1_R_GGTATCTGTTATTTTCATCCATCATATAGTC EDS1_R1437_AAGCATGATCCGCACTCGGATCCGT TCTTTAAATGTCGATGGTAGTTTGC EDS1_F1621_CTACCATCGACATTTAAAGAACGGAT CCGAGTGCGGATCAT
EDS1Δ358-383	EDS1_R1071_CTATCCATGCTAGTTTCTTTAAAAAGT ACTGTCTGCCTCTTGTGCTC EDS1_F1150_AGTGAGCACAAGAGGCAGACAGTACT TTTTAAAGAACTAGCATGGATAGAAG
EDS1Δ310-383	EDS1_R930_TTGGCTTGTATTCATCTTCTATCCA ATAGCTATGATGATCTCTGATACTTCGA EDS1_F1168_AAGTATCAGAGATCATCATAGCTAT TGGATAGAAGATGAATACAAGCCA
EDS1Δ310-350	EDS1_R930m3_CGTA CTGTCTGCCTCTTGTGCT TGGAATTAGAGACCATTCTTGTTCATC EDS1_F1051_AAGAATGGTCTCTAATTCCA AGCACAAGAGGCAGACAGT
EDS1Δ331-350	EDS1_R990_CGTA CTGTCTGCCTCTTGTGCT CTTCTTTCCCATCGACTGTACC EDS1_F1051m5_TGGTACAGTCGATGGGAAAGAAG AGCACAAGAGGCAGACAGT
EDS1Δ310-330	EDS1_R930m4_CTCCATCCAAATGATTAACAA TGGAATTAGAGACCATTCTTGTTCATC EDS1_F991m4_AAGAATGGTCTCTAATTCCA TTGTTTAATCATTTGGATGGAGAAAAC

EDS1_1-120	GW_R_TGTGAACACTATCTGTTTTCTACTCCT
EDS1_1-200	GW_R_GACAAAGTTCACAAAGAACCG
EDS1_1-273 (eds1-2)	GW_R_CACTGCAACCAATCTCTTCTC
EDS1_1-300	GW_R_GTGATCCTCTTTCAAAGCAACTTG
EDS1_310-424	GW_F310_ATGCCATTCGAAGTATCAGAGATCATCATAG GW_R_CTTGACGTTTGCTTTGAAGTCATTC
EDS1_310-383	GW_R_AAACCTCTCTTGCTCGATCAC
EDS1_310-350	GW_R_CACTCCAAGGTCATTGAGCGTAG
EDS1_310-330	GW_R_CTTCTTTCCCATCGACTGTACC
EDS1_357-623	GW_F_ATGTACGTTCAAGCTGCATTAGAGG
EDS1_385-623	GW_F_ATGTTAAAGAACTAGCATGGATAGAAG
EDS1_410-623	GW_F_ATGTCCTTCAAAGTTTCAAATGAAGAGAATG
EDS1_440-623	GW_F_ATGAAGAAATGTCAACTTCCAGATGAG
EDS1_480-623	GW_F_ATGGAAGACACAGGGCCGTAC
EDS1_542-623	GW_F_ATGTCCGAGTGCGGATCATGC
EDS1_542-593	GW_R_TGAACCCTCCAGAAATATTTTCCTTATC

qPCR

eEF-1 α	F: GATTGCCACACCTCTCACATTGC R: GGTCTTCTTGTCCACGCTCTT
WRKY18	F: CGACATACGAAGGGACGCATAAC R: CCAGATATAGCAGCAGCAAGAGC
WRKY40	F: CTCACTATTGGCGTTACTCGTATGC R: CCTCTCGGTTATGTTGCTCTTGTT
WRKY38	F: AAGATCAGGATCAGGAGAAGGAAAGC R: CCAGATGACGATGAAGGAGGATAAGAG
WRKY46	F: GCTTCAACCAAGACAAGAATCCT R: TCCAGCAGTGACCATCATCAATAGA
WRKY54	F: GCAAGAGCAAGAGAACAACACCAG R: GCACAATATGAGTCGAAGAAGGAGGAA
WRKY70	F: ACACCATCTCCGTTCTTGATACCTT R: TCTGGACTTGCTTTGTTGCCTTG
DND1	F: CTAACCCAGCCCGAACCAGATT R: CACAACCAACGACAAGCGACTC
ERECTA	F: AATACGGTTCTCTTCTTCTTCTATCCT R: CACACCTCTCCAGACACAATAATCC
PR1	F: GCGAGAAGGCTAACTACAACACTAC R: CCTGCATATGATGCTCCTTATTGA
PR2	F: CTTAGCCTCACCACCAATGTTG R: CGGAATCTGACACCATCTCTGTA
PR5	F: CCACAGACTTCACTCTAAGGAACAAT R: GTAACCATCTACGAGGCTCACATC

WRKY53	F: TGACTGGTTCAATCCAACGGTCG R: GCCGCAAACTGATGGAAGCATAAC
WRKY46	F: GCTTCAACCAAGACAAGAACATCCT R: TCCAGCAGTGACCATCATCAATAGA
SARD1	F: GGAGAAGTAAGTGTGACGGTGAGAA R: TGTTGATGTGGCGAGAGGAGAG
PAD4	F: TCAATCTTCTCCGCCGTCATTCC R: AGTGTCCGTACCTCTGATGTTCT
ICS1	F: CTAACGAGAACGGAAACGGAAAC R: TACCACCATAGGCACGAATCAG
PBS3	F: CGTCCTGAATTAGCAGACACTATTGAAG R: CTCCGAAGAACCGTAAGTTGTTGAA

ChIP-qPCR

Actin2	F: GTAACATTGTGCTCAGTGGTGGAA R: CCTGGACCTGCCTCATCATACT
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PR1 amplicon genomic loci relative to TSS

F1 (+336/+606)	F: GCGAGAAGGCTAACTACAACACTAC R: CCTGCATATGATGCTCCTTATTGA
F2 (-235/-7)	F: GGCAAAGCTACCGATACGAAAC R: GGGTTCGTAAACATCGCTTATATAGAG
F3 (-574/-300)	F: GCATGAAACACTAAGAAACAAATAATTCTTG R: TGTATATAGTTGTTTCATGTCATTCAGTTG
F4 (-765/-578)	F: ACAAAGTGTATAACAATGTCAATCGGTG R: TGAGTATCTCTATCACTCTTGCCTATG
F5 (-2417/-2204)	F: ATGTTTGAGGTTGAGTACGATGGAC R: TGTATTCTCTTTGCTTCTCCACAAAG

PR2 amplicon genomic loci relative to TSS

R1 (-320/-143)	F: CCATAATTGAACTTGGCTTGTGGGT R: GAATCGCCCAAACCAATTTGGTT
R2 (-401/-295)	F: GAACCATCCATGGCACCTTAAAC R: GACCCACAAGCCAAGTTCAATTATG
R3 (-531/-405)	F: CGAACGTGTTTTACAAGTTGAGAC R: GAACCACTGTGAACACAAATCATGC
R4 (-706/-576)	F: GCACCTTGTGTTTTACTGAACCAAAT R: GTCGTTTTCCCCCTTCATGATG

PBS3 amplicon genomic loci relative to ATG code

S1 (-255/-135)	F: CACTTCACACGCTAAGGTCAGT R: ATGGATTTGGCAGTTCGTGAGT
S2 (-561/-383)	F: CCATGAAGCCTTAGATCGATGTCAAAGT R: CCATGGAAAATGTTTTCTTTTGTCGGC
S3 (-890/-753)	F: CGACAAGATGATACTTGCTGAACAATTC

S4 (-1229/-1062)	R: GCAGTATACTAGTACCATGCAAAATTGTG F: CTTTCATCACACGACTCACTCAACT
S5 (-1345/-1230)	R: TACACAAAGACCAGACCACAACAAG F: ACCAATCTGGAGAAAAGATACGAGAC
S6 (-2182/-2068)	R: TGATGGAGACTAGAAATTACAAACGAAGTTTC F: ATGGACGCTACTTAAATCGACACG
S7 (-2341/-2222)	R: TTCTAGCGAGAAAGGACAAGCCAT F: GTACATTAATAATAAGATCCCCTTTGATTTATGG
S8 (-2843/-2697)	R: TCGGGCCGTATAAGACATTAC F: GTCAGAAGATGGAGACTACTTGGGAAG R: TGGAGGGATATTGCCCAGAAATTC

EDS1 amplicon genomic loci relative to TSS

E1 (+312/+599)	F: AGCTTCTGTGGAAATGGCTGTG R: ACAGACGCCTTTCGAGCAAG
E2 (-167/-39)	F: ACCGACACGTGGAAAGCTAAG R: TACCCAAAATACCCCATCATGAGACC
E3 (-355/-239)	F: AACCGAGAGTGACATCTCAAACC R: TTTGGTCGGAAAGTTTTCTCTGTGT
E4 (-562/-418)	F: CTTGTAGATTATCCGGTTTACTTTCCGG R: GAATGAGGAGATTTGTTCGCTTGGAG
E5 (-622/-609)	F: TTCTAGTCCGGTTCATGTA ACTTC R: ACTAACTACACCTTCTTGCTGG
E6 (-1281/-1184)	F: ACACATGCATGTCCTGATTCTTTTCG R: CCCATCTTTGTCACTCTTGATCAGTCT

Genotyping

GFP	F: AGGGTGAAGGTGATGCAACATACG R: TCCAGCAGGACCATGTGATCG
eGFP	F: AAGCTGACCCTGAAGTTCATCTGC R: TCCAGCAGGACCATGTGATCG
<i>eds1-2</i>	F: GACCCTTTCTAGTTTCCTTGAGCTAAGTC R: CGTCTGTGATCCATTCTCCAAGC WT, 1065 bp; <i>eds1-2</i> , 222 bp.
<i>npr1-2</i> (CAPS) Restriction enzyme	F: GGATGATTTCTACAGCGACGCT R: GTAACCATAGCTTAATGCAGATGGTG FspI: WT, 270 bp and 139 bp; <i>npr1-2</i> , 409 bp.
<i>npr1-3</i> (dCAPS) Restriction enzyme	F: GGCCGACTATGTGTAGAAATACTAGCG R: TGAGACGGTCAGGCTCGAGG HhaI: WT, 319 bp and 27 bp; <i>npr1-3</i> , 346 bp.

Supplementary Table 2. Recombinant DNA

Plasmid name	Source
pDEST-GBKT7-NPR1	This paper
pDEST-GBKT7-EDS1	This paper
pDEST-GBKT7-PAD4	This paper
pDEST-GBKT7-PAD4_1-340	This paper
pDEST-GBKT7-SAG101	This paper
pDEST-GBKT7-CDK8	This paper
pDEST-GBKT7-EDS1_Δ480-540	This paper
pDEST-GBKT7-EDS1_Δ358-383	This paper
pDEST-GBKT7-EDS1_Δ310-383	This paper
pDEST-GBKT7-EDS1_1-200	This paper
pDEST-GBKT7-EDS1_1-273 (eds1-2)	This paper
pDEST-GBKT7-EDS1_1-300	This paper
pDEST-GBKT7-EDS1_310-424	This paper
pDEST-GBKT7-EDS1_310-383	This paper
pDEST-GBKT7-EDS1_Δ310-350	This paper
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pDEST-GBKT7-TGA3	This paper
pDEST-GBKT7-EDS1_1-120	This paper
pDEST-GBKT7-EDS1_1-350	This paper
pDEST-GBKT7-EDS1_1-384	This paper
pDEST-GBKT7-EDS1_1-424	This paper
pDEST-GBKT7-EDS1_357-623	This paper
pDEST-GBKT7-EDS1_385-623	This paper
pDEST-GBKT7-EDS1_410-623	This paper
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pDEST-GBKT7-EDS1_480-623	This paper
pDEST-GBKT7-EDS1_542-623	This paper
pDEST-GBKT7-EDS1_542-593	This paper
pDEST-GADT7-SAG101	This paper
pDEST-GADT7-PAD4	This paper
pDEST-GADT7-EDS1	This paper
pDEST-GADT7-NPR1	(ref. ³⁴)
pDEST-GADT7-GUS	(ref. ³⁴)
pDEST-GADT7-EDS1	This paper
pDEST-GADT7-NPR1_1-195 (BTB)	This paper
pDEST-GADT7-NPR1_Δ60-150	This paper
pDEST-GADT7-NPR1_1-500	This paper
pDEST-GADT7-NPR1_375-593	This paper
pDEST-GADT7-NPR1ANK	This paper
pDEST-GADT7-NPR1C521A	(ref. ³⁴)
pDEST-GADT7-NPR1C529A	(ref. ³⁴)

pDEST-GADT7-npr1-2	This paper
pDEST-GADT7-nim1-2	(ref. ³⁴)
pDEST-GADT7-npr1-1	(ref. ³⁴)
pDEST-GADT7-npr1-5	(ref. ³⁴)
pDEST15-GST-EDS1	This paper
pDEST15-GST-PAD4	This paper
pDEST15-GST-GUS	This paper
pET32a-Trx-His ₆ -NPR1	(ref. ³⁴)
pMDC43-nVenus-EDS1	This paper
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pMDC43-cCFP-PAD4	This paper
pMDC43-cCFP-EDS1	This paper
pCAMBIA1300-EDS1-nLUC	This paper
pCAMBIA1300-cLUC-NPR1	This paper
pCAMBIA1300-cLUC	(ref. ⁹¹)
pCAMBIA1300-nLUC	(ref. ⁹¹)
pCAMBIA1300-cLUC-RAR1	(ref. ⁹¹)
pCAMBIA1300-SGT1b-nLUC	(ref. ⁹¹)
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