

Supplementary Figure 1. Characterization of the *cbnac1* mutant and CBNAC overexpressing transgenic lines.

(A) Schematic representation of the *CBNAC* gene and the T-DNA insertion in the *cbnac1* mutant. Black boxes indicate exons, and thin lines indicate introns or untranslated regions. The position of T-DNA insertion is indicated by a reverse triangle.

(**B**) RNA gel blot analysis of the *cbnac1* mutant was performed as in Fig. S1 on total RNA isolated from leaves of wild type (WT) and *cbnac1* plants 6 h after treatment with 1 mM SA.

(C) CBNAC expression in *35S:Flag-CBNAC* transgenic plants. Protein samples were prepared from leaves of four-week-old wild type (WT) and T3 homozygous progeny of transgenic plants (*35S:Flag-CBNAC*) on SDS-PAGE and detected with anti-Flag antibody. Rubisco was used as a loading control.



Supplementary Figure 2. CBNAC cDNA rescues the cbnac1 disease phenotype.

(A) CBNAC expression is restored to wild type (WT) levels in *cbnac1/CBNAC* transgenic lines. Shown is a gel blot analysis total RNA isolated from leaves treated for 6 h with 1 mM SA using a *CBNAC* fragment as probe.

(B) Growth of *Pst*DC3000 in leaf tissues of wild-type and *cbnac1*/CBNAC plants at 0 and 3 dpi was measured after treatment as in Figure 1. Mean bacterial densities \pm SE were calculated from six to eight replicate plants. Significant differences according to Student's *t* test (P < 0.05) are indicated by unique letters. The experiment was repeated three times with similar results.

(C) qRT-PCR analysis of *PR1* expression in leaves inoculated as above was performed at 0 and 12 hpi. *PR1* expression levels were normalized using the expression level of *Tubulin 2*, and then expressed relative to the expression level in WT at 12hpi which is arbitrarily set at 100. Mean expression values \pm SE were calculated from the results of three independent experiments.



Supplementary Figure 3. EMSA showing coarse mapping of CBNAC-binding sites on the *PR1* promoter.

(A) Diagram of the *PR1* promoter showing the DNA fragments used for EMSA. The positions of individual fragments relative to the translation start codon are shown above the line.

(**B**) EMSA showing CBNAC binding to different fragments of the *PR1* promoter. The locations of DNA fragments used for EMSA are shown in (**A**).



Supplementary Figure 4. EMSA showing intermediate mapping of CBNAC-binding sites on the *PR1* promoter.

(A) Diagram of the overlapping DNA oligonucleotides used in EMSA. The positions of individual oligonucleotides relative to the translation start codon are shown.

(**B**) EMSA showing CBNAC binding to different fragments of the *PR1* promoter. The locations of DNA oligonucleotides used for EMSA are shown in (**A**).

Supplementary Figure 5. EMSA showing fine mapping of CBNAC-binding sites on the *PR1* promoter.

(A) Diagram of DNA oligonucleotides used in EMSA. The positions of individual oligonucleotides relative to the start codon are shown.

(**B**) EMSA of CBNAC binding to different regions of the *PR1* promoter. The radioactive probes were incubated in the presence of bacterially expressed GST-CBNAC or GST alone. The locations of DNA oligonucleotides used for EMSA are shown in (**A**).

(C) ChIP assays of *in vivo* DNA binding by CBNAC. *In vivo* DNA–protein complexes in chromatin were covalently cross-linked by formaldehyde and recovered from leaves of wild-type (WT) and *35S:Flag-CBNAC* (Flag-CBNAC) transgenic plants. Chromatin was then sonicated to yield a population of soluble fragments and incubated with BSA as a negative control (No Ab) or with specific antibodies against CBNAC (α -Flag Ab). Target *PR1* promoter sequences (E0, E3, E4, E5 and E6) were detected by PCR using specific primers. Input refers to PCR reactions performed on samples prior to immunoprecipitation.

Supplementary Figure 6. Identification of the CBNAC domain required for interaction with SNI1.

(A) Schematic diagram of full-length and different fragments of CBNAC that were fused to the Gal4 DNA binding domain (BD). The putative CaMBD in the C terminus and the NAC domain in the N terminus are represented with a *black* and *gray* box, respectively. Numbers indicate positions of amino acid residues. Interaction of each construct with SNI1 is depicted as positive (+) or none (-).

(**B**) Yeast two-hybrid results of the Gal4 activation domain (AD) -full-length SNI1 fusion construct with the various BD-CBNAC fusion constructs shown in (**A**). Different combinations of plasmids are represented by numbers 1 to 6.

Supplementary Figure 7. CBNAC binds to the *PR-1* promoter in SA-induced leaves. Shown is a CHIP assay of CBNAC from leaves of wild-type (WT) and *35S:Flag-CBNAC* (Flag-CBNAC) transgenic plants treated for 12 h with 1 mM SA. *In vivo* DNA–protein complexes in chromatin were covalently cross-linked by formaldehyde. Chromatin was then recovered, sonicated to yield a population of soluble fragments, and incubated with BSA as a negative control (No Ab) or with specific antibodies against CBNAC (α -Flag Ab). Target *PR1* promoter sequence (E0) was detected by PCR using specific primers. Input refers to PCR reactions performed on samples prior to immunoprecipitation.

Supplementary Figure 8. JA-induced gene expression pattern is unaltered in the *cbnac1* mutant. (A) and (B) Leaves of four-week-old *Arabidopsis* plants (Col-0) and *cbnac1* were treated with 0.1 mM JA. Leaf collection, RNA isolation, and RNA gel blot analysis were performed as in Figure 1 except that *PDF1.2* (A) and *LOX2* (B) fragments were used as probe.

Supplementary Figure 9. Interaction of CBNAC with SNI1 is mitigated by pathogen treatment. LCI assay was performed as in Figure 3*B*. Shown are luminescence images (upper panel) and quantitative luminescence measurements (lower panel) depicting luciferase activity in inoculated leaves at 48 hpi. *Pst*DC3000 ($OD_{600} = 0.001$ in 10 mM MgCl₂) was treated after 24 h as Agro infiltration. Symbols: '+' indicates *Pst*DC3000 treatment, whereas '-' indicates no *Pst*DC3000 treatment.

Supplementary Table 1. Primer	sequences used for the	e confirmation of the	e mutant (<i>cbnac1</i> ,
cbnac1sni1), RT-PCR, and qPCB	R.		

Primer Name	Primer Sequence (5'-3')	Primer Name	Primer Sequence (5'-3')
PR1-qPCR-F	GTGGGTTAGCGAGAAGGCTA	CBNAC-S	CTCGAGTGGATTCTCTCGACTGAT
PR1-qPCR-R	ACTTTGGCACATCCGAGTCT	F1	GAATCCGAGAAACCATCAGATTAT
Tubulin2-qPCR-F	ulin2-qPCR-F CCAACAACGTGAAATCGACAG		CTCGAGTGAAAATCTCTCTCCTTT
Tubulin2-qPCR-R TCTTGGTATTGCTGGTACTCT		SNI1-S1	TTTTGCAACAGCTGATTC
T-DNA GTGATGGTTCACGTAGTTGGCCATCG		SNI1-S2	CTCGAGTCAAGCTTTTGCCTTGAGTAC

Supplementary Table 2. Primer sequences used for mapping of the CBNAC-binding promoter region of the *PR1* gene.

Primer Name	Primer Sequence (5'-3')	Primer Name	Primer Sequence (5'-3')
EO	F : TAATAATGCTTAGTTATAAATTACT	E5 2	F:ACTTAAATTAGAATCATGAA
EU	R : AGTAATTTATAACTAAGCATTATTA	E3-2	R : AGTTGTTTCATGTCATTCAG
E1	F:ACAGATTCATATCAGG	E5 2	F:ATATACAATGTTTCTTAATA
	R : AACTCTAGGTGACCGATCTA	E2-2	R : AGTATATATGTAAGTATACC
E2	F:AGTAGATCGGTCACCTAGA	E4 1	F:AGGGTATACTTACATATATA
	R:AGAGAAACAGTCAATAGATC	E0-1	R:TAATATTGTTTCGTATCGGT
БЭ	F:TCTCAATGGGTGATCTATTG	E6 3	F:GGAAAAATGTGTGTAAGGAC
ES	R:CTCTATCACTCTTGCCTATG	E0-2	R:AATTGTCCAAATGAA
E4	F:CCATAGGCAAGAGTGATAGA	E0 1 1	F:TAATAATGCTTAGTTATAAATTACT
E4	R :CAAAATGTTTTGAAGATATC	E0-1-1	R : AGTAATTTATAACTAAGCATTATTA
E.	F:AAGATATCTTCAAAACATTT	E0 1 2	F:ATTTATTCATGCTAAACTATTTCTCGTAACTATTA
ES	R :AGTATATATGTAAGTATACC	E0-1-2	R : TAATAGTTACGAGAAATAGTTTAGCATGAATAAAT
E	F:AGGGTATACTTACATATATA	E0 4 1	F :CTTTAAAAAATCAATTTTTCTGATTCGGAGGGAGTATA
Eo	R :AATTGTCCAAATGAA	EU-4-1	R : TATACTCCCTCCGAATCAGAAAAATTGATTTTTTAAAG
E0 1	F : TAATAATGCTTAGTTATAAAT	E0 4 2	F:TGTTATTGCTTAGAATCACAGATTC
E0-1	R : TAATAGTTACGAGAAATAGTT	E0-4-2	R :GAATCTGTGATTCTAAGCAATAACA
F 0.2	F:ACCAATAGTAATTCATCAAAT	F2 1 1	F:TCTCAATGGGTGATCTATTG
E0-2	R : TATGAATTTCAAGAATCAATT	E3-1-1	R:CAATAGATCACCCATTGAGA
F 0.2	F : ACCTTTTAATATTGATTGATA	E3-1-2	F:CTATTGACTGTTTCTCTACGTCACTATT
E0-5	R :TTTTTTTGTATTAAAAAGATT		R : AATAGTGACGTAGAGAAACAGTCAATAG
E0.4	F:CTTTAAAAAATCAATTTTTCT	F2 1 2	F:CACTATTTTACTTACGTCAT
E0-4	R :GAATCTGTGATTCTAAGCAAT	E3-1-3	R :ATGACGTAAGTAAAATAGTG
E2 1	F:TCTCAATGGGTGATCTATTG	F411	F: ATACTCATATGCATGAAACACTAAGAAAC
E3-1	R :ATGACGTAAGTAAAATAGTG	E4-1-1	R:GTTTCTTAGTGTTTCATGCATATGAGTAT
E3-2	F :AGATGTGGCGGCATATATTC	E412	F : AAATAATTCTTGACTTTTTT
	R :CTCTATCACTCTTGCCTATG	Ľ4-1-2	R :AAAAAGTCAAGAATTATTT
E4-1	F :CCATAGGCAAGAGTGATAGA	E5 2 1	F :ATATACAATGTTTCTTAATAAACTTCATTT
	R : AAAAAAGTCAAGAATTATTT	E5-3-1	R :AAATGAAGTTTATTAAGAAACATTGTATAT
E4-2	F:TCTTTTATTTGAAAATTGAC	E5 2 2	F : AGGGTATACTTACATATATA
	R :GCAATTAAGATTATATTTAC	E5-5-2	R : TATATATGTAAGTATACCCT
E4-3	F:CAAACTGTCCGATACGATTT	F(1 1	F: AAAAAAATATATCAACAATGGCAAAGCT
	R :CAAAATGTTTTGAAGATATC	E0-1-1	R:ACGTTTGCCATTGTTGATATATTTTTTT
E5-1	F:AAGATATCTTCAAAACATTT	E6-1-2	F:ACCGATACGAAACAATATTA
	R:CTTCTATTTCAAATTTGAAT		R:TAATATTGTTTCGTATCGGT