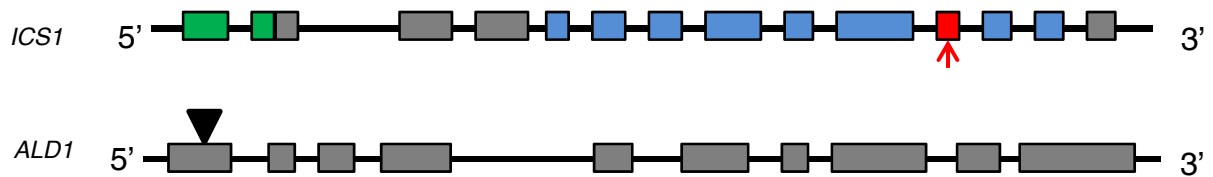
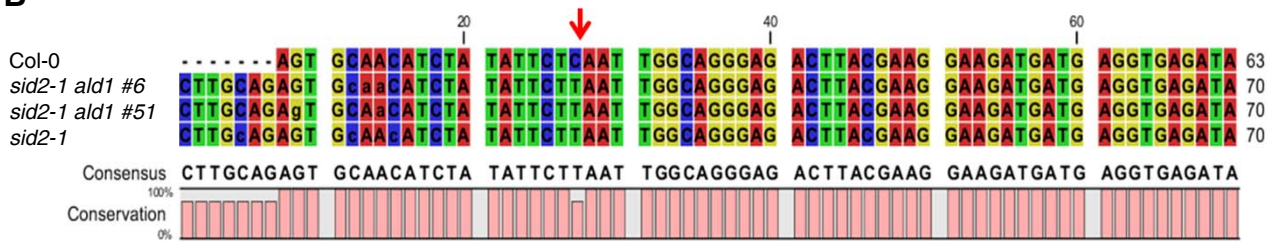
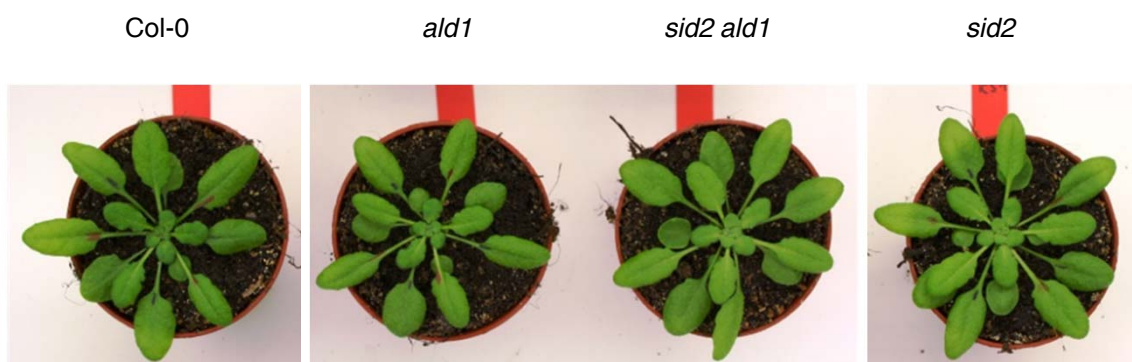


Supplemental Figure 1. Identification of the *sid2 ald1* (*sid2-1 ald1*) double mutant.

(A) PCR analysis of genomic DNA isolated from Col-0 and F2 *sid2 ald1* candidate lines to screen for *ALD1* T-DNA insertion a: Left genomic primer (LP) and right genomic primer (RP) primer, b: LP and left border T-DNA insertion primer (LB), c: RP and LB primer pair. M = 100 bp marker. Among others, lines #2, #4, #6, #13, #19, #23 were identified as homozygous for the insertion.

(B) Gradient PCR (57 - 67°C) using genomic DNA of Col-0 and *sid2-1*, employing the ICS1-FV primer site-specific for the wild-type *ICS1* sequence but not for the point-mutated *sid2-1* variant (in combination with ICS1-RV). 63.8°C was identified as annealing temperature specifically amplifying Col-0 but not *sid2-1* genomic DNA. H₂O served as a negative control. M = 100 bp marker.

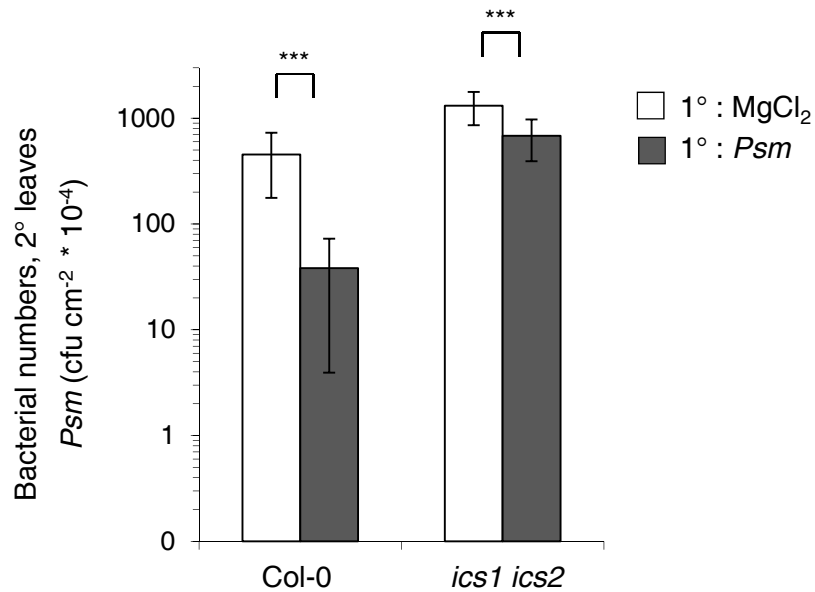
(C) PCR analysis of genomic DNA isolated from Col-0, *sid2* and preselected F2 *sid2 ald1* candidate lines. The ICS1-FV/ICS1-RV primer pair and an annealing temperature of 64°C were used. M = 100 bp marker. Lines #6 and #51 (not shown) were identified as *sid2 ald1* double mutant lines homozygous for both mutations and further characterized (Suppl. Fig. 2; Fig. 1).

A**B****C****Supplemental Figure 2. Characterization of the *sid2 ald1* double mutant.**

(A) Schematic representation of the *ICS1* and *ALD1* genes (5'→3'). *ICS1* contains a putative plastid transit sequence (green) and a chorismate-binding domain (blue). The location of mutations in *sid2-2* and *sid2-1* are indicated with a red box and a red arrow, respectively (modified according to Wildermuth et al., 2001). In the scheme of the *ALD1* locus, the position of the T-DNA insertion (black triangle) of SALK_007673 is indicated. Boxes represent exons and spaces represent spliced introns.

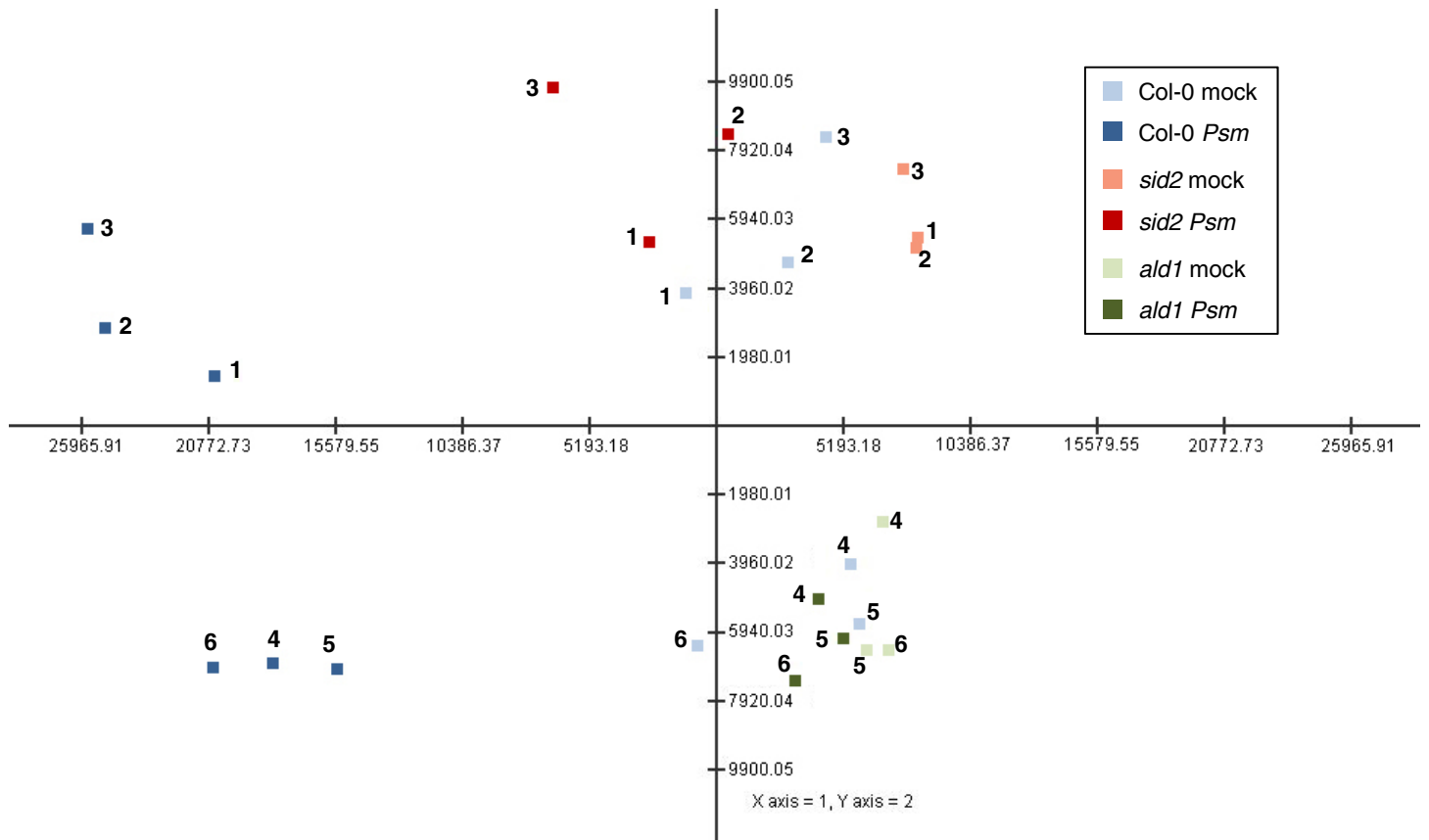
(B) Multiple sequence alignment of Col-0, the positively PCR-tested F2 *sid2 ald1* candidates #6 (Suppl. Fig. 1) and #51, and *sid2-1*.

(C) Growth phenotypes of naïve 5-week-old Col-0, *ald1*, *sid2 ald1* and *sid2* plants. The *sid2 ald1* double mutant plants did not show any obvious morphological phenotype that distinguished them from wild-type, *ald1*, or *sid2* plants.



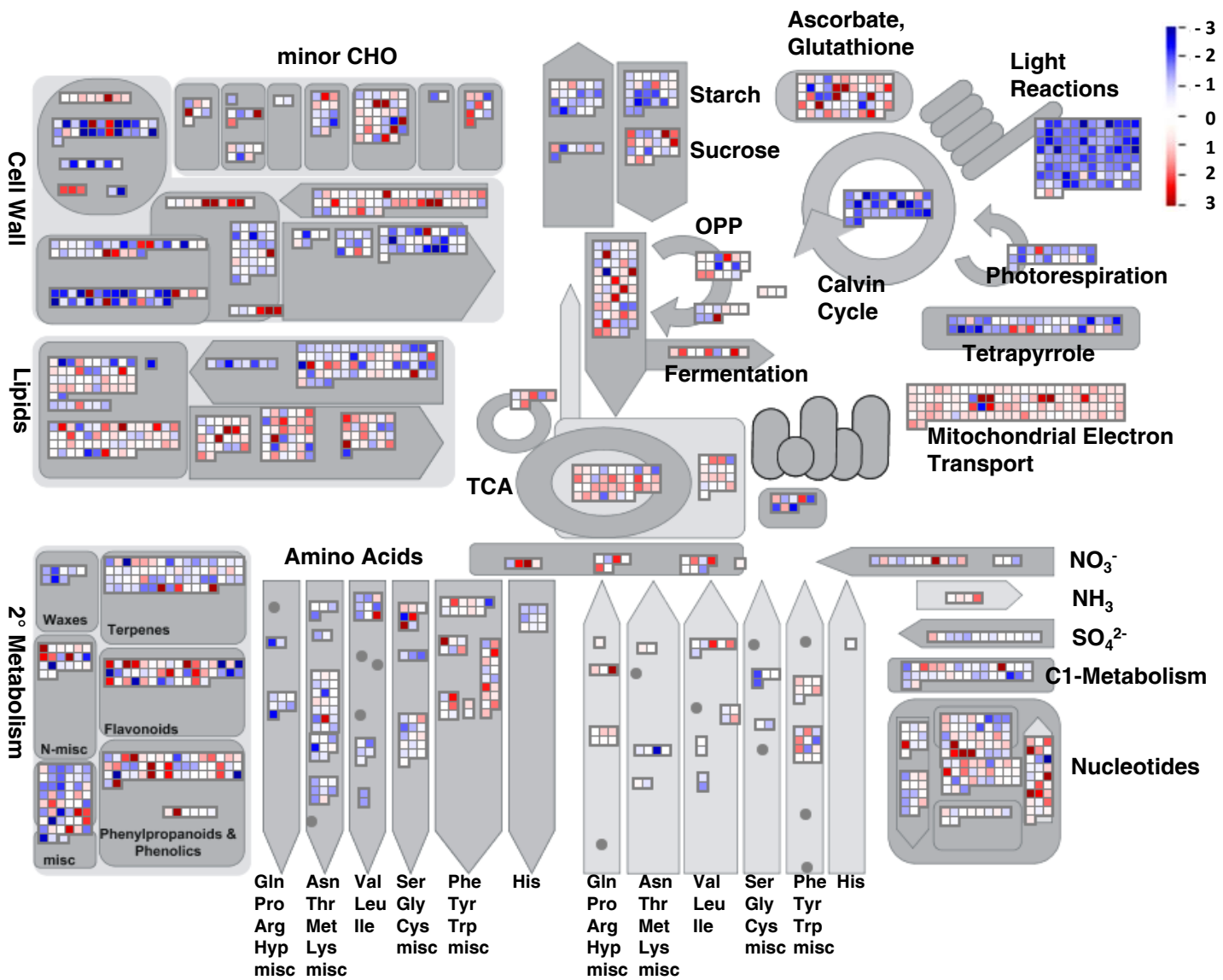
Supplemental Figure 3. Systemic acquired resistance assay with *Col-0* and *ics1 ics2* plants.

Lower (1°) leaves were infiltrated with either 10 mM MgCl_2 or *Psm* ($\text{OD}_{600} = 0.005$) to induce SAR, and two days later, three upper leaves (2°) were challenge-infected with *Psm* ($\text{OD}_{600} = 0.001$). Bacterial growth in upper leaves was assessed 3 days post 2° leaf-inoculation ($n \geq 7$). Asterisks denote statistically significant differences between *Psm*-pre-treated and mock-control samples (***: $P < 0.001$; two-tailed *t* test). Seeds of *ics1 ics2* were sterilized and germinated on full MS medium containing 2 % sucrose (pH 5.7) before the seedlings were transferred to soil (Garcion et al., 2008).



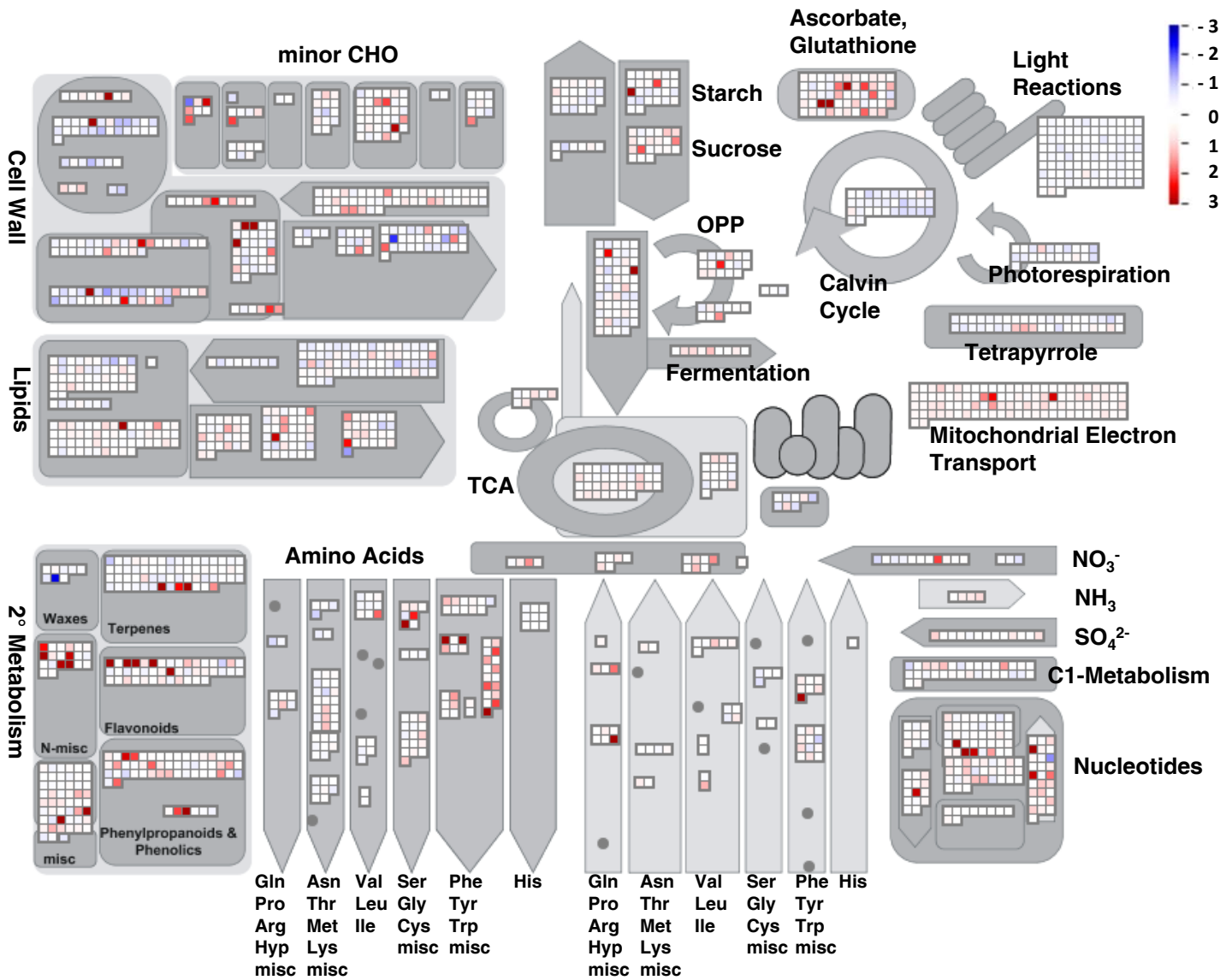
Supplemental Figure 4. Principle component analysis of the normalized transcriptome data; 1st dimension 58.0 % of variation; 2nd dimension 15.7 % of variation.

The principle component analysis of all samples indicates that 58.0 % of the variation is treatment variation which separates the *Psm*-inoculated from the mock-infiltrated samples. The wild-type has the farthest separation followed by *sid2*. The separation in *ald1* is virtually non-existent. The second dimension is environmental variation between the two sets of independent experiments (SAR experiments 1 to 3 vs. SAR experiments 4 to 6) and comprises only 15.7 % of variation.

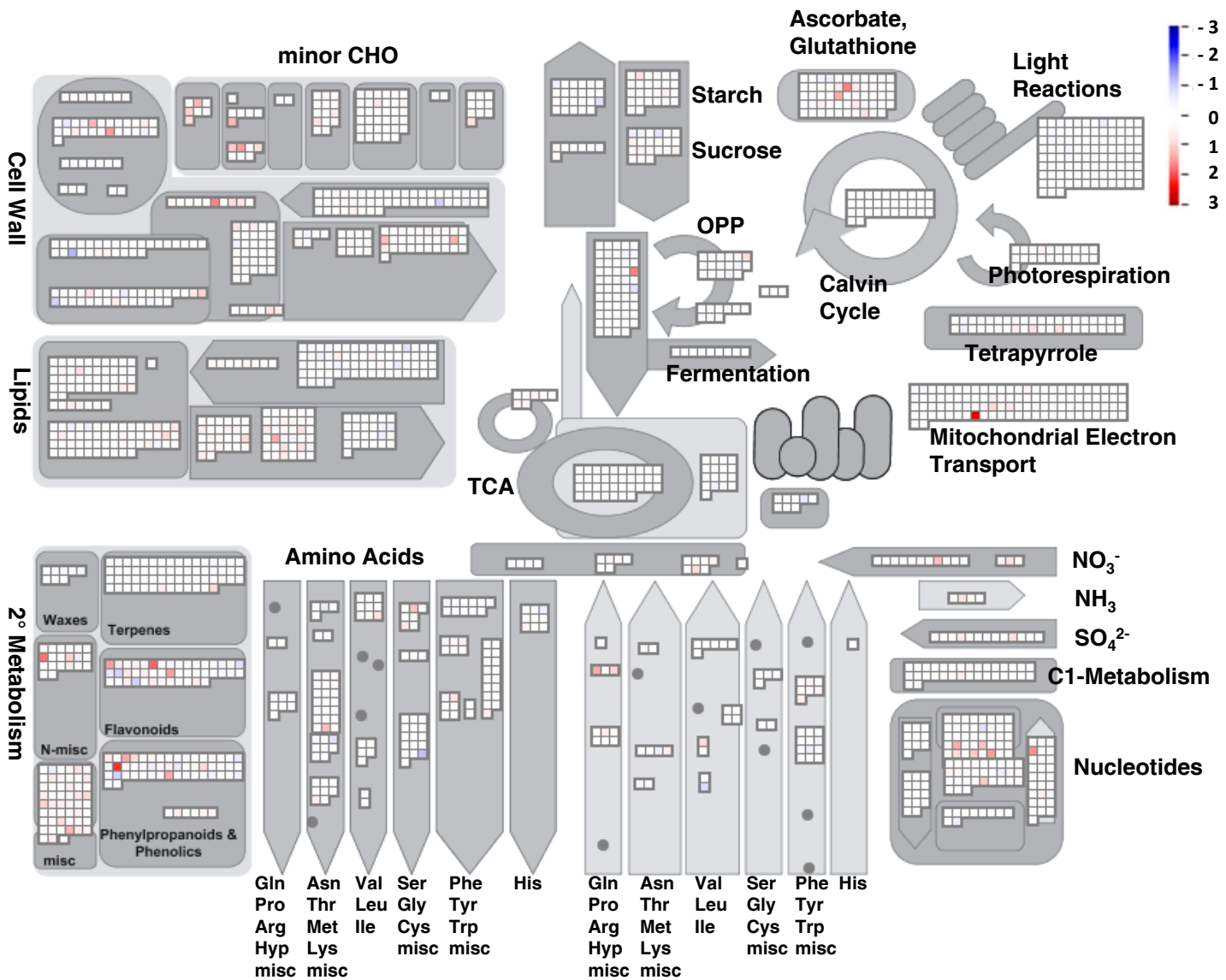


Supplemental Figure 5. MapMan visualization: the transcriptional SAR response in Col-0.

Overview of the changes in metabolism-related gene expression in distal (2°) leaves of Col-0 wild-type plants inoculated in 1° leaves with *Psm* (*P*) compared to Mock (*M*). Heat map representation indicates log₂ *P/M*-fold changes. Red (blue): Up-regulated (down-regulated) upon *Psm* inoculation.

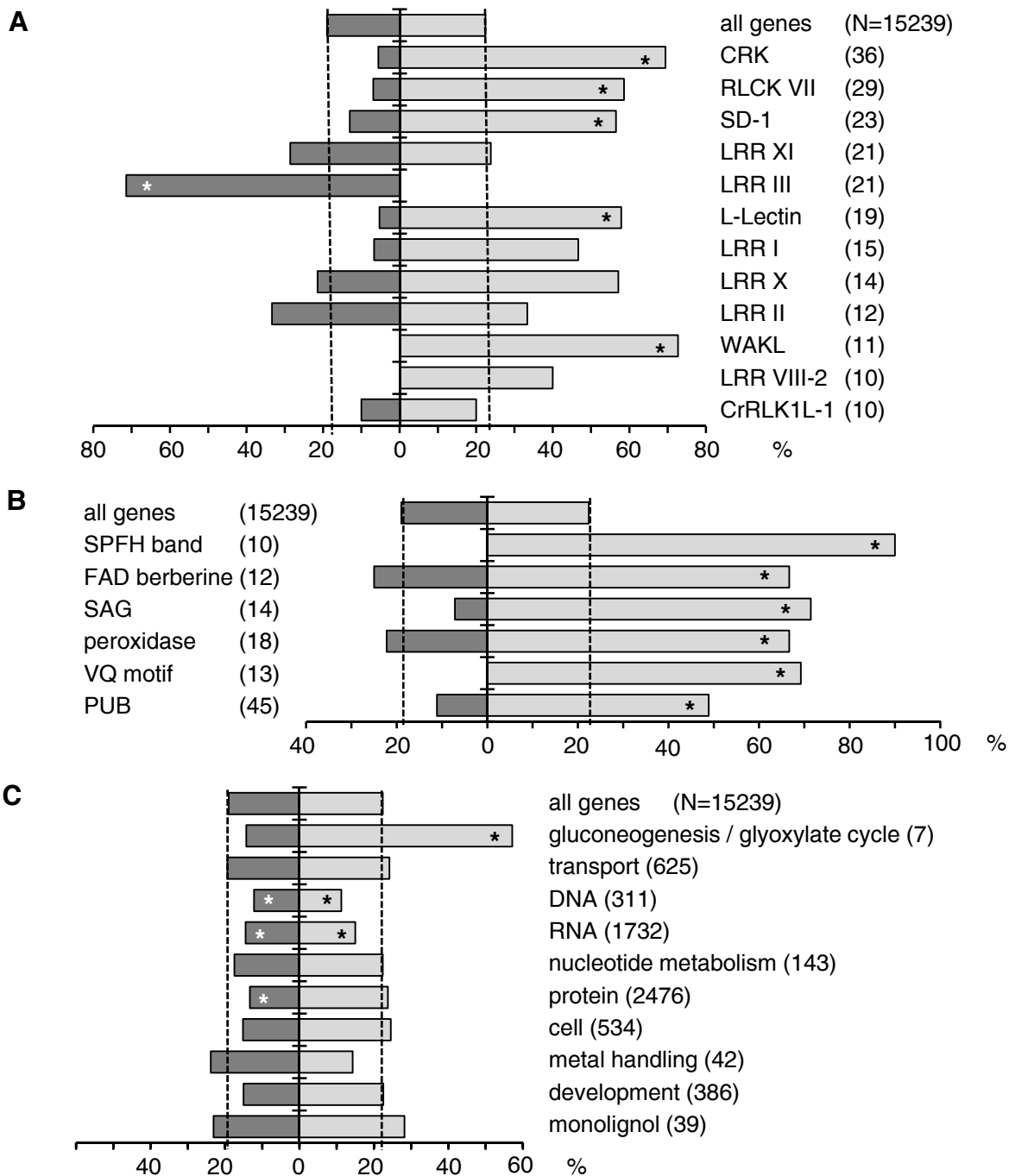


Supplemental Figure 6. MapMan visualization: a diminished transcriptional SAR response exists in *sid2*. Overview of the changes in metabolism-related gene expression in distal (2°) leaves of *sid2* plants inoculated in 1° leaves with *Psm* (*P*) compared to Mock (*M*). Heat map representation indicates log₂ *P/M*-fold changes. Red (blue): Up-regulated (down-regulated) upon *Psm* inoculation.



Supplemental Figure 7. MapMan visualization: the transcriptional SAR response is absent in *ald1*.

Overview of the changes in metabolism-related gene expression in distal (2°) leaves of *ald1* plants inoculated in 1° leaves with *Psm* (*P*) compared to Mock (*M*). Heat map representation indicates log₂ P/M-fold changes. Red (blue): Up-regulated (down-regulated) upon *Psm* inoculation.



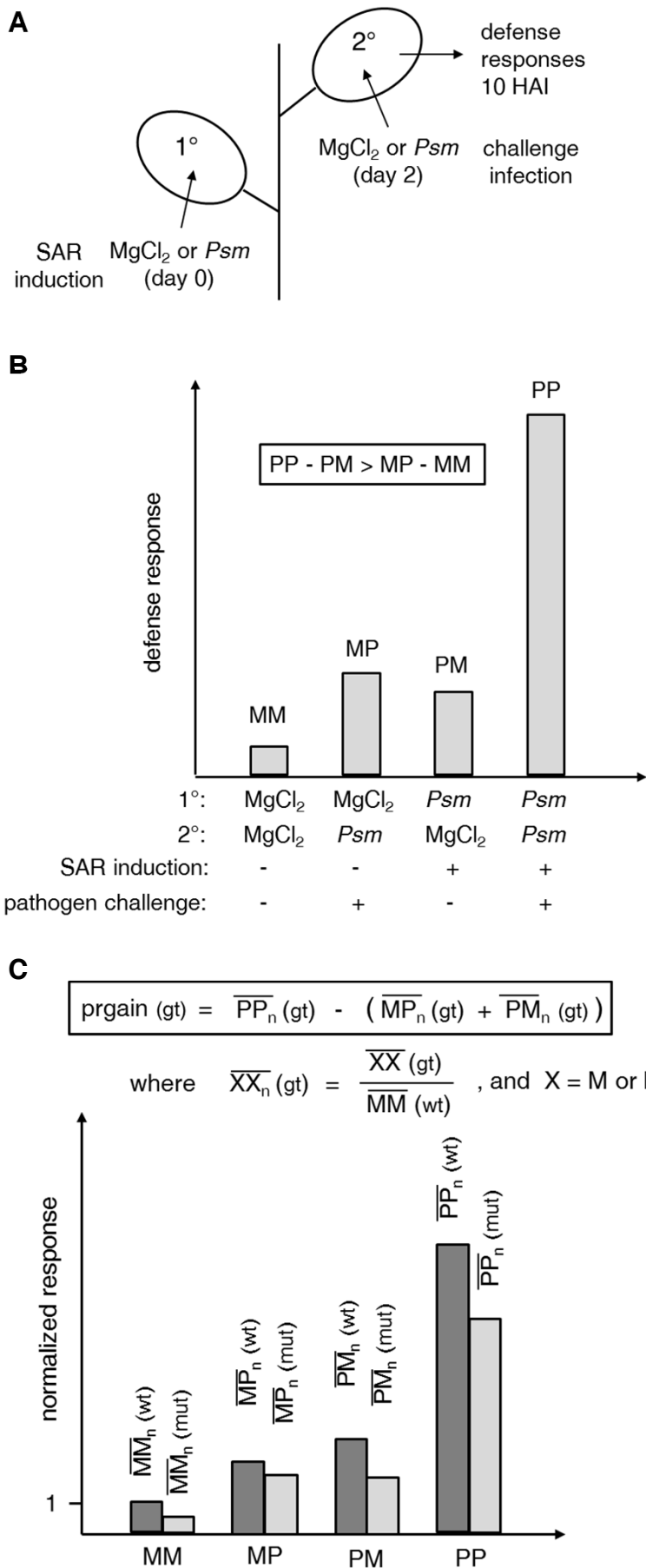
Supplemental Figure 8. Percentage of SAR⁺ and SAR⁻ genes in additional gene classes/families.

(A) Major subclasses of receptor-like protein kinases. Classification according to Shiu et al. Plant Cell (2004) 16: 1220-34. Only subclasses consisting of more than 10 genes were considered. CRK: cystein-rich protein kinases; RLCK: receptor-like cytoplasmic kinases; LRR: leucine rich repeat kinases; WAKL: wall-associated kinases.

(B) Further gene families enriched in SAR⁺ genes. SPFH: stomatin/prohibitin/flotillin/HflK/C domain-containing genes; SAG: senescence-associated genes; FAD berberine: FAD berberine-type genes; VQ-motif: VQ motif-containing genes; PUB: plant U-box gene family.

(C) Other MapMan categories and gene families. Peroxidase: class III peroxidase gene family, Raes et al. Plant Physiol (2003) 133: 1051-71; monolignol: monolignol biosynthesis gene families, Tognolli et al. Gene (2002) 288: 129-38 (<http://www.arabidopsis.org/>).

Dashed vertical lines illustrate the percentage of SAR⁺ and SAR⁻ genes in the analyzed gene set (15239 genes). The number of genes in each category is given in brackets. Asterisks on right (left) bars indicate significant enrichment or depletion of gene categories in SAR⁺ (SAR⁻) genes (Fisher's exact test, $P < 0.01$).



Supplemental Figure 9.

SAR-associated defense priming – assay and definition.

(A) Experimental setup of the SAR priming assay.

At day 0, a SAR-inductive *Psm*- or a mock-control (MgCl₂)-treatment was performed in 1° leaves. This was followed by a *Psm*-challenge or a mock-treatment of 2° leaves 48 h later. Defense responses (gene expression, metabolite analyses) in 2° leaves were assessed 10 h after the second treatment. All four possible combinations were compared: 1°-mock / 2°-mock (control state), 1°-*Psm* / 2°-mock (SAR induction, no pathogen challenge), 1°-mock / 2°-*Psm* (no SAR induction, pathogen challenge), and 1°-*Psm* / 2°-*Psm* (SAR induction and pathogen challenge).

(B) Definition of priming.

A particular defense response was defined as “primed”, if the differences between the (1°-*Psm* / 2°-*Psm*)- and the (1°-*Psm* / 2°-mock)-values were significantly larger than the differences between the (1°-mock / 2°-*Psm*)- and the (1°-mock / 2°-mock)-values, as determined by a two-sided Mann-Whitney U test ($\alpha = 0.005$). Analogous definitions were used to assess the priming of pathogen- and SA-responses after exogenous Pip-treatment (Pip/*Psm* – Pip/mock > H₂O/*Psm* – H₂O/mock) and (Pip/SA – Pip/mock > H₂O/SA – H₂O/mock), respectively.

(C) Response gain due to priming.

To estimate quantitative differences between genotypes in the extent of priming of a response, we calculated the parameter “response gain due to priming” (prgain). The prgain-value reflects the gain of a response in a genotype if priming is activated.

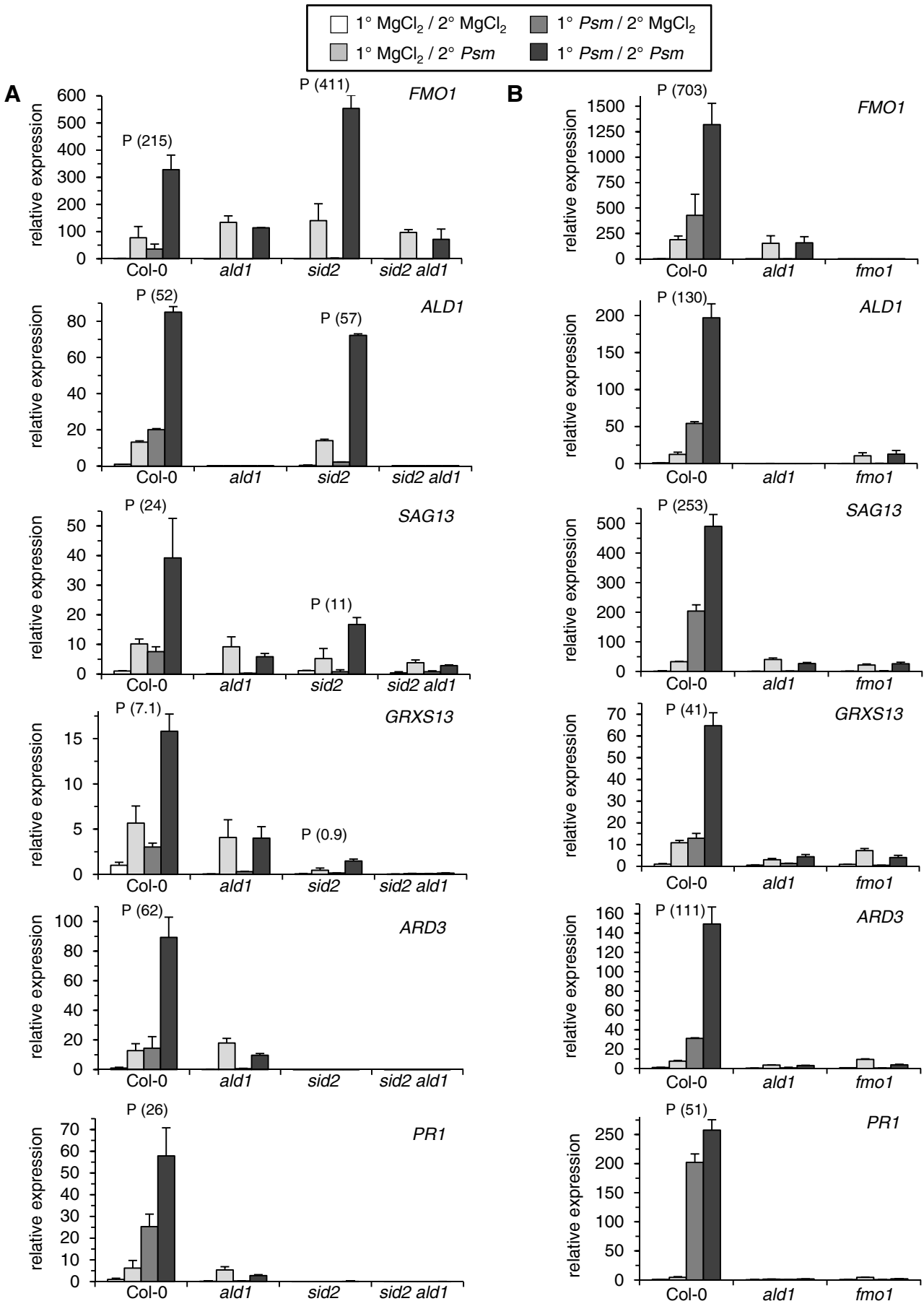
$\overline{XX}(\text{gt})$: mean of response values of a genotype (gt); X = M (Mock) or P (*Psm*).

$\overline{XX}_n(\text{gt})$: $\overline{XX}(\text{gt})$ normalized to $\overline{MM}(\text{wt})$, the mean of the wild-type (wt) mock/mock- values.

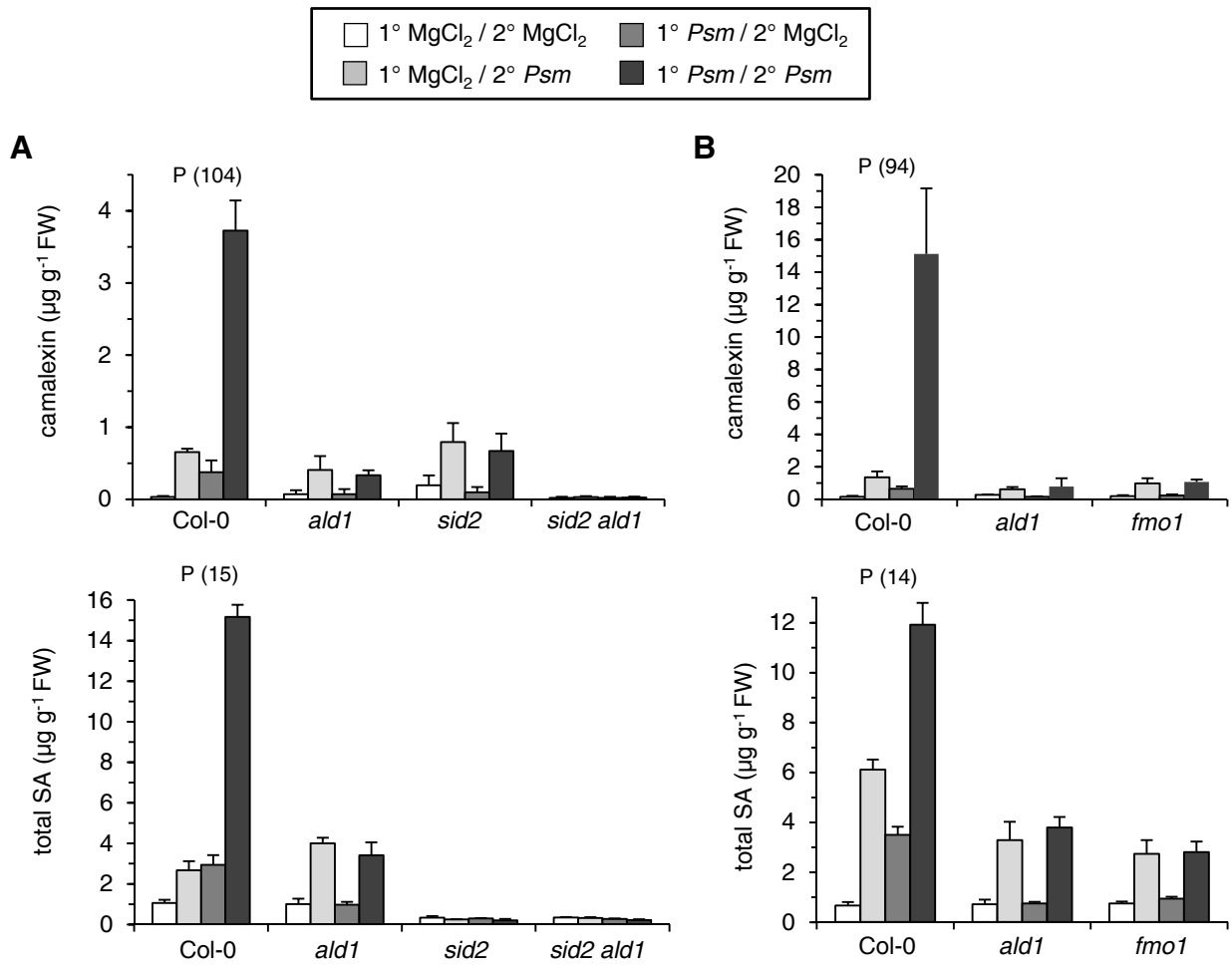
mut: mutant.

Note that the values of all genotypes are normalized to $\overline{MM}(\text{wt})$. Thus, $\overline{MM}_n(\text{wt})$ of a dataset is always equal to 1.

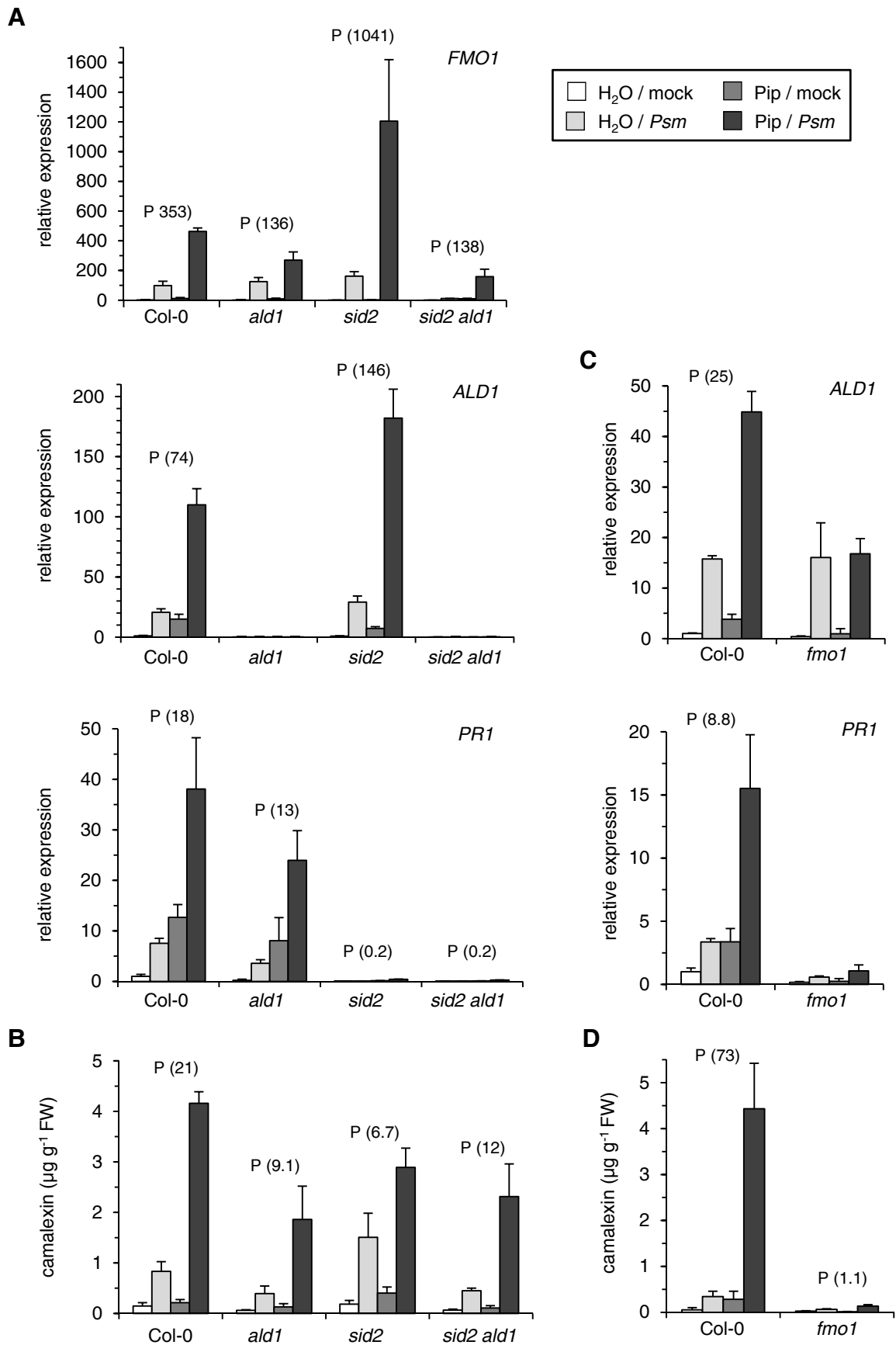
The above description relates to biological SAR. The prgain-values for Pip-induced priming of *Psm*- or SA-responses were calculated analogously.



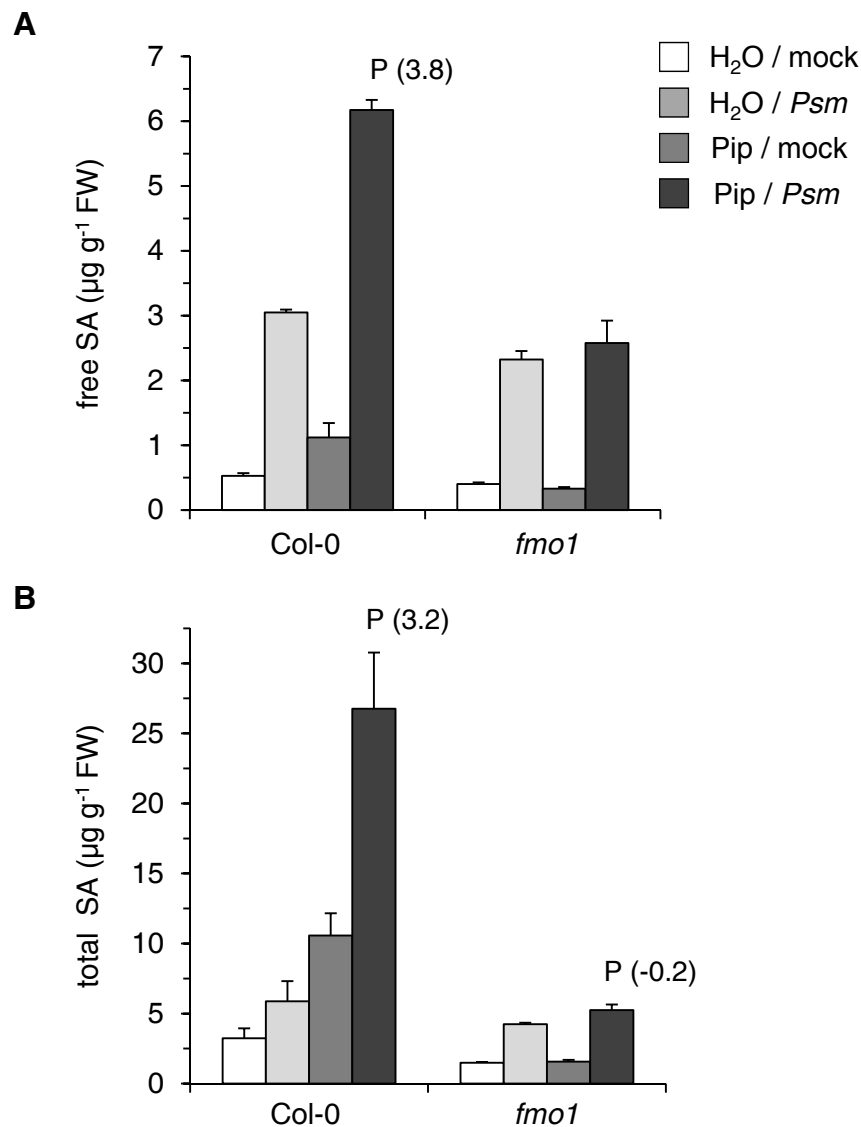
Supplemental Figure 10. Graphs of Figure 6 with a linear scale for the y-axes instead of a log-scale.



Supplemental Figure 11. Graphs of Figure 7 with a linear scale for the y-axes instead of a log-scale.



Supplemental Figure 12. Graphs of Figure 8 with a linear scale for the y-axes instead of a log-scale.

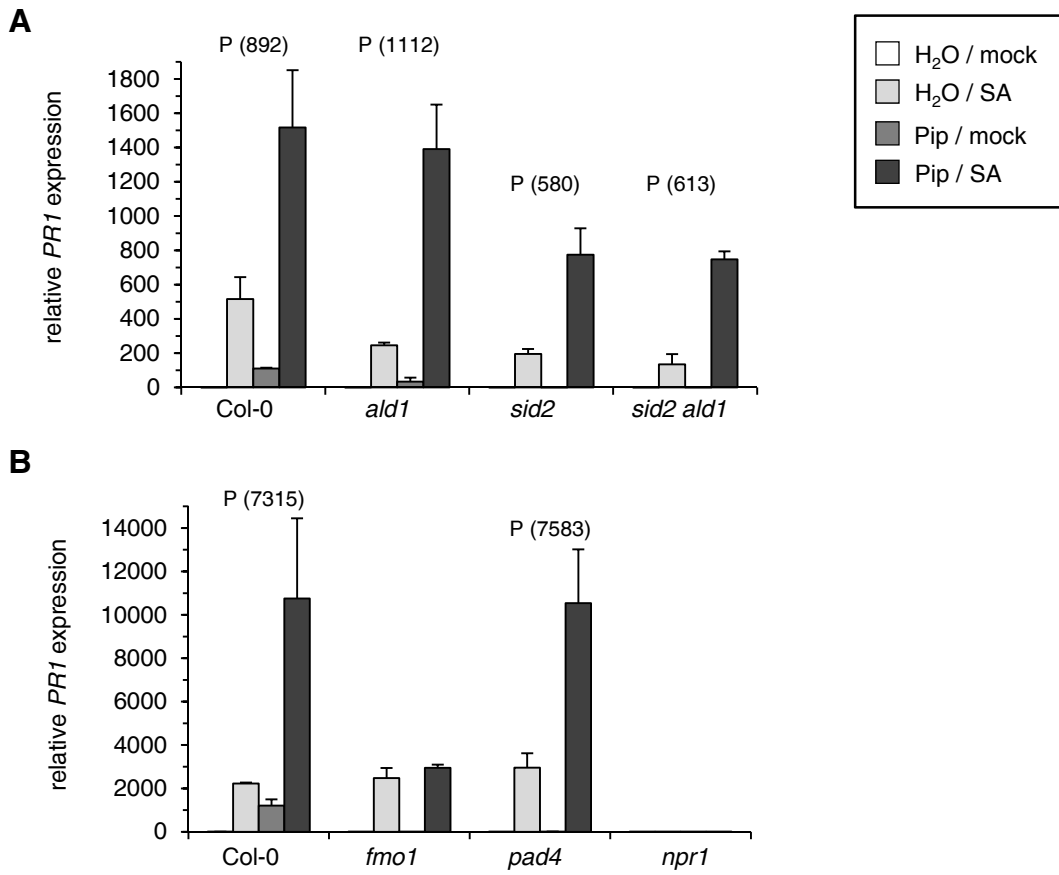


Supplemental Figure 13. Pip-induced priming of salicylic acid biosynthesis requires functional *FMO1*.

(A) free salicylic acid.

(B) total salicylic acid (sum of free and glycosidic SA).

Plants were supplied with 10 ml of 1 mM Pip (\equiv dose of 10 μmol) or with 10 ml of H₂O (control treatment) via the root system, and leaves challenge-inoculated with *Psm* or mock-infiltrated one day later. Defense responses in leaves were assessed 10 h after the challenge treatment. Values represent the mean \pm SD of three biological replicates from different plants. Each biological replicate consists of six leaves from two plants. A “P” above the bars representing Pip/*Psm*-treatments indicates defense priming, as assessed in analogy to SAR priming (see Supplemental Figure 9B). As a measure of the gain of a response due to priming, we calculated the prgain (“response gain due to priming”) for each genotype with activated priming according to the formula given in Supplemental Figure 9C online. Prgain-values are given in brackets behind the priming indicator “P” and allow estimates about quantitative differences of the strength of priming between genotypes. The higher the prgain-value, the stronger the priming.



Supplemental Figure 14. Graphs of Figures 9A and 9B with a linear scale for the y-axes instead of a log-scale.

				mean expression value						fold-change (log ₂)			
	AGI	name	function	Col-0		<i>sid2</i>		<i>ald1</i>		Col-0	<i>sid2</i>	<i>ald1</i>	
				M	P	M	P	M	P	P/M	P/M	P/M	
A	At1g57630	-	TIR-NBS-LRR protein	6	256	1	56	2	3	5.1*	4.9*	0.4	
	At1g66090	-	TIR-NBS protein	10	212	4	21	9	26	4.3*	2.1*	1.4	
	At4g11170	<i>RMG1</i>	TIR-NBS-LRR protein	1	34	0	2	0	1	4.1*	1.4*	0.7	
	At5g41750	-	TIR-NBS-LRR protein	24	293	6	27	10	67	3.5*	2.1*	2.6	
	At5g46520	<i>VICTR</i>	TIR-NBS-LRR protein	8	57	4	12	6	9	2.7*	1.5	0.5	
B	At4g23210	<i>CRK13</i>	cysteine-rich protein kinase	7	174	2	26	0	2	4.5*	3.1*	1.1	
	At4g04500	<i>CRK37</i>	cysteine-rich protein kinase	12	275	3	35	1	1	4.4*	3.1*	0.2	
	At1g51890	-	receptor-like protein kinase	6	221	0	13	2	3	5.0*	3.4*	0.7	
	At1g51800	<i>IOS1</i>	receptor-like protein kinase	7	88	1	6	1	2	3.5*	2.0*	0.3	
	At4g33430	<i>BAK1</i>	receptor-like protein kinase	32	140	23	34	25	26	2.1*	0.6	0.0	
	At5g20480	<i>EFR</i>	receptor-like protein kinase	6	28	6	10	2	1	2.0*	0.6	-0.6	
	At3g21630	<i>CERK1</i>	receptor-like protein kinase	33	112	21	36	20	24	1.7*	0.7	0.2	
	At4g46330	<i>FLS2</i>	receptor-like protein kinase	19	17	11	7	17	19	-0.1	-0.6	0.2	
	C	At1g01560	<i>MPK11</i>	MAP kinase	7	193	1	16	1	3	4.5*	3.1*	0.7
		At3g45640	<i>MPK3</i>	MAP kinase	109	601	70	152	75	138	2.5*	1.1	0.9
At2g43790		<i>MPK6</i>	MAP kinase	43	118	34	41	43	50	1.5*	0.3	0.2	
At4g29810		<i>MKK2</i>	MAP kinase kinase	65	363	41	112	27	33	2.5*	1.4*	0.3	
At4g01370		<i>MKK4</i>	MAP kinase kinase	68	277	57	111	41	43	2.0*	0.9	0.1	
At3g46930		<i>Raf43</i>	MAP kinase kinase kinase	4	37	3	8	2	6	3.0*	1.1	1.1	
At5g66850		<i>MAPKKK5</i>	MAP kinase kinase kinase	32	178	23	28	29	34	2.4*	0.2	0.2	
D		At1g76040	<i>CPK29</i>	Ca ²⁺ -dependent protein kinase	14	295	6	47	5	13	4.3*	2.8*	1.2
	At1g18890	<i>CPK1</i>	Ca ²⁺ -dependent protein kinase	33	256	21	48	22	31	2.9*	1.2	0.4	
	At4g35310	<i>CPK5</i>	Ca ²⁺ -dependent protein kinase	23	126	13	30	19	28	2.4*	1.2	0.5	
E	At1g80840	<i>WRKY40</i>	WRKY transcription factor	4	169	2	11	3	46	5.0*	2.1*	3.4	
	At5g13080	<i>WRKY75</i>	WRKY transcription factor	1	50	0	2	1	0	4.9*	1.4*	-0.1	
	At5g64810	<i>WRKY51</i>	WRKY transcription factor	3	107	0	4	1	1	4.8*	2.2*	0.1	
	At2g46400	<i>WRKY46</i>	WRKY transcription factor	18	419	5	79	6	23	4.4*	3.7*	1.7	
	At4g23810	<i>WRKY53</i>	WRKY transcription factor	8	162	1	8	2	23	4.2*	2.0*	2.9	
	At4g31800	<i>WRKY18</i>	WRKY transcription factor	15	233	6	15	4	16	3.8*	1.2	1.7	
	At5g46350	<i>WRKY8</i>	WRKY transcription factor	3	46	1	5	2	2	3.7*	1.5*	0.2	
F	At3g44350	<i>NAC061</i>	NAC transcription factor	2	69	0	6	0	1	4.5*	2.3*	0.2	
	At5g22380	<i>NAC090</i>	NAC transcription factor	4	86	0	10	0	4	4.1*	3.1*	1.6	
	At2g43000	<i>NAC042</i>	NAC transcription factor	3	57	0	4	1	1	3.9*	1.8*	-0.1	
G	At1g22070	<i>TGA3</i>	TGA transcription factor	29	150	27	60	12	16	2.3*	1.1	0.4	
	At5g06960	<i>TGA5</i>	TGA transcription factor	10	50	7	14	11	13	2.2*	0.9	0.2	
	At5g65210	<i>TGA1</i>	TGA transcription factor	12	40	12	14	8	9	1.6*	0.3	0.3	
H	At1g64280	<i>NPR1</i>	transcriptional coactivator	39	121	31	55	20	26	1.6*	0.8	0.3	
	At5g45110	<i>NPR3</i>	NPR1-like protein 3	25	234	16	31	6	14	3.2*	1.0	1.0	
	At4g19660	<i>NPR4</i>	NPR1-like protein 4	32	85	26	36	6	6	1.4*	0.5	0.0	

Supplemental Figure 15. The transcriptional SAR response: activation of multiple stages of defense signaling. Expression values of selected genes.

Selected genes among **(A)** resistance proteins, **(B)** receptor-like protein kinases, **(C)** MAP kinase cascade members, **(D)** calcium-dependent protein kinases, **(E)** WRKY transcription factors, **(F)** NAC transcription factors, **(G)** TGA transcription factors, and **(H)** NPR1 and paralogues.

The mean of the expression values of the RNA-seq analyses are depicted. Samples originate from distal leaves of *Psm* (*P*)-inoculated or mock (*M*)-treated Col-0, *sid2*, or *ald1* plants at 48 HAI. Log₂-transformed *P/M*-ratios (fold-changes) are depicted, and asterisks indicate significant changes (FDR < 0.01). The log₂ *P/M*-ratios are highlighted according to their values as follows:

log ₂ <i>P/M</i>	≥ 5.0	4.9 to 4.0	3.9 to 3.0	2.9 to 2.0	1.9 to 1.0	0.9 to -0.9	-1.0 to -1.9	-2.0 to -2.9	≤ -3.0
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(A)	Experiment	Treatment1	Treatment2	Phenotype
A	Mock	Mock	Mock	1.56
A	Mock	Mock	Mock	0.46
A	Mock	Mock	Mock	0.97
A	Mock	Psm	Psm	27.21
A	Mock	Psm	Psm	147.60
A	Mock	Psm	Psm	57.77
A	Psm	Mock	Mock	57.09
A	Psm	Mock	Mock	37.48
A	Psm	Mock	Mock	11.89
A	Psm	Psm	Psm	303.08
A	Psm	Psm	Psm	402.47
A	Psm	Psm	Psm	278.32
B	Mock	Mock	Mock	1.56
B	Mock	Mock	Mock	1.14
B	Mock	Mock	Mock	0.31
B	Mock	Psm	Psm	239.44
B	Mock	Psm	Psm	152.13
B	Mock	Psm	Psm	176.21
B	Psm	Mock	Mock	241.64
B	Psm	Mock	Mock	328.27
B	Psm	Mock	Mock	716.20
B	Psm	Psm	Psm	1238.10
B	Psm	Psm	Psm	1116.76
B	Psm	Psm	Psm	1606.59
C	Mock	Mock	Mock	0.94
C	Mock	Mock	Mock	0.90
C	Mock	Mock	Mock	1.16
C	Mock	Psm	Psm	137.05
C	Mock	Psm	Psm	186.87
C	Mock	Psm	Psm	234.39
C	Psm	Mock	Mock	17.70
C	Psm	Mock	Mock	28.02
C	Psm	Mock	Mock	19.39
C	Psm	Psm	Psm	740.93
C	Psm	Psm	Psm	455.76
C	Psm	Psm	Psm	531.36

ANOVA results	SumSq	Df	F value	Pr(>F)
Treatment1	1270647	1	93.0036	9.93*10 ⁻¹⁰
Treatment2	1197332	1	87.6374	1.76*10 ⁻⁰⁹
Experiment	923292	2	33.7897	1.05*10 ⁻⁰⁷
Treatment1:Treatment2	415170	1	30.3879	1.14*10 ⁻⁰⁵
Treatment1:Experiment	739373	2	27.0588	7.07*10 ⁻⁰⁷
Treatment2:Experiment	189701	2	6.9425	0.004178
Treatment1:Treatment2:Experiment	93249	2	3.4126	0.04962
Residuals	327896	24		

Supplemental Table 1. Linear model-based analysis of the SAR-associated priming response in Col-0 plants to estimate treatment and experimental effect terms.

(A) Phenotype: relative *FMO1* transcript levels.

An analysis of variance (ANOVA) was performed with the depicted data (“object1”) from three independent experiments (A, B, and C), each consisting of three biological replicate samples per treatment type, using the R statistical package and the command:

“Anova(lm(Phenotype~Treatment1+Treatment2+Experiment+Treatment1*Treatment2+Treatment1*Experiment+Treatment2*Experiment+Treatment1*Treatment2*Experiment, data=object1),type=2)”.

SAR priming was assessed as described in Fig. 6 and Supplemental Fig. 11.

Treatment1: effect term for treatment of 1° leaves (Mock or *Psm*); Treatment2: effect term for the subsequent treatment of 2° leaves (Mock or *Psm*).

Treatment1*Treatment2: effect term for the interaction of treatments (“Priming”).

Experiment: Term reflecting variation between experiments; Experiment*Treatmentx: Term reflecting experimental impact on effect of treatment x; Experiment*Treatment1*Treatment2: Term reflecting experimental influence on treatment interaction.

SumSq: type II-sum of squares. Df: degrees of freedom. Pr(>F): P-value associated with a corresponding F value.

(B)	Experiment	Treatment1	Treatment2	Phenotype			
A	Mock	Mock	1.2				
A	Mock	Mock	0.7				
A	Mock	Mock	1.1				
A	Mock	Psm	4.1				
A	Mock	Psm	18.9				
A	Mock	Psm	16.6				
A	Psm	Mock	27.7				
A	Psm	Mock	16.7				
A	Psm	Mock	15.8				
A	Psm	Psm	65.6				
A	Psm	Psm	65.2				
A	Psm	Psm	124.3				
B	Mock	Mock	1.01				
B	Mock	Mock	1.19				
B	Mock	Mock	0.80				
B	Mock	Psm	11.24				
B	Mock	Psm	9.55				
B	Mock	Psm	16.59				
B	Psm	Mock	56.98				
B	Psm	Mock	51.22				
B	Psm	Mock	54.44				
B	Psm	Psm	220.84				
B	Psm	Psm	174.70				
B	Psm	Psm	195.29				
C	Mock	Mock	0.54				
C	Mock	Mock	1.12				
C	Mock	Mock	1.35				
C	Mock	Psm	29.01				
C	Mock	Psm	58.07				
C	Mock	Psm	52.72				
C	Psm	Mock	50.11				
C	Psm	Mock	44.16				
C	Psm	Mock	39.46				
C	Psm	Psm	292.02				
C	Psm	Psm	343.28				
C	Psm	Psm	237.15				
ANOVA results				SumSq	Df	F value	Pr(>F)
Treatment1				94985	1	232.523	7.59*10 ⁻¹⁴
Treatment2				68426	1	167.506	2.57*10 ⁻¹²
Experiment				26172	2	32.035	1.68*10 ⁻⁰⁷
Treatment1:Treatment2				36996	1	90.566	1.28*10 ⁻⁰⁹
Treatment1:Experiment				15705	2	19.223	1.04*10 ⁻⁰⁵
Treatment2:Experiment				17744	2	21.719	4.13*10 ⁻⁰⁶
Treatment1:Treatment2:Experiment				8210	2	10.049	0.0006753
Residuals				9804	24		

Supplemental Table 1. (B) Phenotype: relative *ALD1* transcript levels.

(C)	Experiment	Treatment1	Treatment2	Phenotype			
A	Mock	Mock	0.91				
A	Mock	Mock	0.94				
A	Mock	Mock	1.14				
A	Mock	Psm	7.20				
A	Mock	Psm	9.80				
A	Mock	Psm	5.94				
A	Psm	Mock	7.69				
A	Psm	Mock	10.54				
A	Psm	Mock	12.34				
A	Psm	Psm	55.75				
A	Psm	Psm	23.14				
A	Psm	Psm	38.79				
B	Mock	Mock	1.17				
B	Mock	Mock	1.03				
B	Mock	Mock	0.80				
B	Mock	Psm	34.68				
B	Mock	Psm	31.41				
B	Mock	Psm	32.70				
B	Psm	Mock	196.09				
B	Psm	Mock	183.71				
B	Psm	Mock	232.66				
B	Psm	Psm	432.95				
B	Psm	Psm	524.47				
B	Psm	Psm	512.16				
C	Mock	Mock	0.71				
C	Mock	Mock	1.58				
C	Mock	Mock	0.70				
C	Mock	Psm	10.85				
C	Mock	Psm	9.45				
C	Mock	Psm	13.98				
C	Psm	Mock	33.79				
C	Psm	Mock	29.51				
C	Psm	Mock	13.73				
C	Psm	Psm	88.83				
C	Psm	Psm	60.36				
C	Psm	Psm	70.05				
ANOVA results				SumSq	Df	F value	Pr(>F)
Treatment1				154917	1	499.78	< 2.2*10 ⁻¹⁶
Treatment2				42262	1	136.344	2.20*10 ⁻¹¹
Experiment				207991	2	335.501	< 2.2*10 ⁻¹⁶
Treatment1:Treatment2				24514	1	79.084	4.62*10 ⁻⁰⁹
Treatment1:Experiment				178702	2	288.257	< 2.2*10 ⁻¹⁶
Treatment2:Experiment				36871	2	59.475	5.02*10 ⁻¹⁰
Treatment1:Treatment2:Experiment				25189	2	40.632	1.97*10 ⁻⁰⁸
Residuals				7439	24		

Supplemental Table 1. (C) Phenotype: relative SAG13 transcript levels.

(D)	Experiment	Treatment1	Treatment2	Phenotype			
A	Mock	Mock	1.28				
A	Mock	Mock	0.53				
A	Mock	Mock	1.19				
A	Mock	Psm	2.99				
A	Mock	Psm	6.92				
A	Mock	Psm	7.07				
A	Psm	Mock	2.40				
A	Psm	Mock	3.42				
A	Psm	Mock	3.24				
A	Psm	Psm	15.50				
A	Psm	Psm	13.44				
A	Psm	Psm	18.54				
B	Mock	Mock	1.33				
B	Mock	Mock	0.77				
B	Mock	Mock	0.91				
B	Mock	Psm	11.09				
B	Mock	Psm	12.01				
B	Mock	Psm	9.45				
B	Psm	Mock	15.07				
B	Psm	Mock	13.83				
B	Psm	Mock	9.79				
B	Psm	Psm	72.00				
B	Psm	Psm	64.85				
B	Psm	Psm	57.37				
C	Mock	Mock	0.79				
C	Mock	Mock	1.31				
C	Mock	Mock	0.90				
C	Mock	Psm	9.33				
C	Mock	Psm	11.88				
C	Mock	Psm	18.04				
C	Psm	Mock	15.35				
C	Psm	Mock	11.82				
C	Psm	Mock	8.37				
C	Psm	Psm	52.59				
C	Psm	Psm	75.30				
C	Psm	Psm	35.84				
ANOVA results				SumSq	Df	F value	Pr(>F)
Treatment1				4245.2	1	101.9153	4.08*10 ⁻¹⁰
Treatment2				4487	1	107.7206	2.37*10 ⁻¹⁰
Experiment				1799.9	2	21.6053	4.30*10 ⁻⁰⁶
Treatment1:Treatment2				1631.8	1	39.1741	1.81*10 ⁻⁰⁶
Treatment1:Experiment				1166.4	2	14.0011	9.34*10 ⁻⁰⁵
Treatment2:Experiment				849.4	2	10.1964	0.0006234
Treatment1:Treatment2:Experiment				445.2	2	5.3443	0.0120307
Residuals				999.7	24		

Supplemental Table 1. (D) Phenotype: relative *GRXS13* transcript levels.

(E)	Experiment	Treatment1	Treatment2	Phenotype			
A	Mock	Mock	1.78				
A	Mock	Mock	0.46				
A	Mock	Mock	0.76				
A	Mock	Psm	6.77				
A	Mock	Psm	18.15				
A	Mock	Psm	13.46				
A	Psm	Mock	10.96				
A	Psm	Mock	25.25				
A	Psm	Mock	7.12				
A	Psm	Psm	94.79				
A	Psm	Psm	70.51				
A	Psm	Psm	102.51				
B	Mock	Mock	1.30				
B	Mock	Mock	0.87				
B	Mock	Mock	0.83				
B	Mock	Psm	8.55				
B	Mock	Psm	6.90				
B	Mock	Psm	7.25				
B	Psm	Mock	30.99				
B	Psm	Mock	31.74				
B	Psm	Mock	30.83				
B	Psm	Psm	174.19				
B	Psm	Psm	138.25				
B	Psm	Psm	135.69				
C	Mock	Mock	1.38				
C	Mock	Mock	0.69				
C	Mock	Mock	0.92				
C	Mock	Psm	6.00				
C	Mock	Psm	5.77				
C	Mock	Psm	10.62				
C	Psm	Mock	41.54				
C	Psm	Mock	28.15				
C	Psm	Mock	22.15				
C	Psm	Psm	168.92				
C	Psm	Psm	139.62				
C	Psm	Psm	89.31				
ANOVA results				SumSq	Df	F value	Pr(>F)
Treatment1				43407	1	200.7128	3.74*10 ⁻¹³
Treatment2				25575	1	118.2605	9.33*10 ⁻¹¹
Experiment				2092	2	4.8378	0.01717
Treatment1:Treatment2				18251	1	84.3928	2.51*10 ⁻⁰⁹
Treatment1:Experiment				2810	2	6.496	0.005562
Treatment2:Experiment				549	2	1.2699	0.299061
Treatment1:Treatment2:Experiment				919	2	2.1249	0.141373
Residuals				5190	24		

Supplemental Table 1. (E) Phenotype: relative *ARD3* transcript levels.

(F)	Experiment	Treatment1	Treatment2	Phenotype			
A	Mock	Mock	0.69				
A	Mock	Mock	0.59				
A	Mock	Mock	1.73				
A	Mock	Psm	2.52				
A	Mock	Psm	10.92				
A	Mock	Psm	5.18				
A	Psm	Mock	25.09				
A	Psm	Mock	32.40				
A	Psm	Mock	18.52				
A	Psm	Psm	58.11				
A	Psm	Psm	42.08				
A	Psm	Psm	73.62				
B	Mock	Mock	1.09				
B	Mock	Mock	0.99				
B	Mock	Mock	0.91				
B	Mock	Psm	6.09				
B	Mock	Psm	3.46				
B	Mock	Psm	4.01				
B	Psm	Mock	222.72				
B	Psm	Mock	193.26				
B	Psm	Mock	190.60				
B	Psm	Psm	259.30				
B	Psm	Psm	278.54				
B	Psm	Psm	235.25				
C	Mock	Mock	1.23				
C	Mock	Mock	0.98				
C	Mock	Mock	0.79				
C	Mock	Psm	7.95				
C	Mock	Psm	4.29				
C	Mock	Psm	7.28				
C	Psm	Mock	106.87				
C	Psm	Mock	59.76				
C	Psm	Mock	90.75				
C	Psm	Psm	192.60				
C	Psm	Psm	261.71				
C	Psm	Psm	243.62				
ANOVA results				SumSq	Df	F value	Pr(>F)
Treatment1				176974	1	715.67	< 2.2*10 ⁻¹⁶
Treatment2				15523	1	62.776	3.74*10 ⁻⁰⁸
Experiment				53865	2	108.912	9.13*10 ⁻¹³
Treatment1:Treatment2				12179	1	49.252	2.95*10 ⁻⁰⁷
Treatment1:Experiment				54714	2	110.63	7.71*10 ⁻¹³
Treatment2:Experiment				5571	2	11.264	0.0003548
Treatment1:Treatment2:Experiment				5393	2	10.905	0.0004275
Residuals				5935	24		

Supplemental Table 1. (F) Phenotype: relative *PR1* transcript levels.

(G)	Experiment	Treatment1	Treatment2	Phenotype			
A	Mock	Mock	0.02				
A	Mock	Mock	0.05				
A	Mock	Mock	0.03				
A	Mock	Psm	0.67				
A	Mock	Psm	0.59				
A	Mock	Psm	0.70				
A	Psm	Mock	0.31				
A	Psm	Mock	0.60				
A	Psm	Mock	0.21				
A	Psm	Psm	3.36				
A	Psm	Psm	3.51				
A	Psm	Psm	4.31				
B	Mock	Mock	0.23				
B	Mock	Mock	0.09				
B	Mock	Mock	0.13				
B	Mock	Psm	1.32				
B	Mock	Psm	1.82				
B	Mock	Psm	0.92				
B	Psm	Mock	0.72				
B	Psm	Mock	0.44				
B	Psm	Mock	0.78				
B	Psm	Psm	9.48				
B	Psm	Psm	18.78				
B	Psm	Psm	17.11				
C	Mock	Mock	0.12				
C	Mock	Mock	0.06				
C	Mock	Mock	0.18				
C	Mock	Psm	0.80				
C	Mock	Psm	1.54				
C	Mock	Psm	0.75				
C	Psm	Mock	0.38				
C	Psm	Mock	0.27				
C	Psm	Mock	0.30				
C	Psm	Psm	5.47				
C	Psm	Psm	7.20				
C	Psm	Psm	7.56				
ANOVA results				SumSq	Df	F value	Pr(>F)
Treatment1				139.122	1	62.808	3.73*10 ⁻⁰⁸
Treatment2				182.115	1	82.218	3.21*10 ⁻⁰⁹
Experiment				62.445	2	14.096	8.94*10 ⁻⁰⁵
Treatment1:Treatment2				115.813	1	52.285	1.80*10 ⁻⁰⁷
Treatment1:Experiment				48.466	2	10.94	0.0004198
Treatment2:Experiment				54.492	2	12.301	0.0002103
Treatment1:Treatment2:Experiment				44.751	2	10.102	0.0006563
Residuals				53.161	24		

Supplemental Table 1. (G) Phenotype: Camalexin levels ($\mu\text{g g}^{-1}$ FW).

(H)	Experiment	Treatment1	Treatment2	Phenotype	
A	Mock	Mock	1.15		
A	Mock	Mock	1.19		
A	Mock	Mock	0.85		
A	Mock	Psm	2.03		
A	Mock	Psm	2.95		
A	Mock	Psm	3.03		
A	Psm	Mock	3.27		
A	Psm	Mock	2.26		
A	Psm	Mock	3.29		
A	Psm	Psm	15.92		
A	Psm	Psm	15.13		
A	Psm	Psm	14.45		
B	Mock	Mock	0.67		
B	Mock	Mock	0.84		
B	Mock	Mock	0.49		
B	Mock	Psm	6.22		
B	Mock	Psm	5.58		
B	Mock	Psm	6.55		
B	Psm	Mock	3.90		
B	Psm	Mock	3.49		
B	Psm	Mock	3.11		
B	Psm	Psm	11.04		
B	Psm	Psm	11.60		
B	Psm	Psm	13.12		
C	Mock	Mock	0.67		
C	Mock	Mock	0.84		
C	Mock	Mock	0.49		
C	Mock	Psm	6.22		
C	Mock	Psm	5.58		
C	Mock	Psm	6.55		
C	Psm	Mock	3.89		
C	Psm	Mock	3.52		
C	Psm	Mock	3.10		
C	Psm	Psm	11.04		
C	Psm	Psm	11.60		
C	Psm	Psm	13.12		
ANOVA results		SumSq	Df	F value	Pr(>F)
Treatment1		250.43	1	681.7015	$< 2.2 \times 10^{-16}$
Treatment2		432.02	1	1175.9988	$< 2.2 \times 10^{-16}$
Experiment		0.07	2	0.0907	0.9136
Treatment1:Treatment2		68.53	1	186.5489	8.20×10^{-13}
Treatment1:Experiment		16.45	2	22.3828	3.27×10^{-06}
Treatment2:Experiment		0	2	0.0008	0.9992
Treatment1:Treatment2:Experiment		29.27	2	39.8436	2.37×10^{-08}
Residuals		8.82	24		

Supplemental Table 1. (H) Phenotype: Total salicylic acid levels ($\mu\text{g g}^{-1}$ FW).

(A)	Experiment	Treatment1	Treatment2	Phenotype
A	A	H2O	Mock	1.05
A	A	H2O	Mock	0.95
A	A	H2O	Mock	1.00
A	A	H2O	Psm	124.84
A	A	H2O	Psm	57.83
A	A	H2O	Psm	112.75
A	A	Pip	Mock	12.76
A	A	Pip	Mock	2.95
A	A	Pip	Mock	19.23
A	A	Pip	Psm	494.69
A	A	Pip	Psm	438.20
A	A	Pip	Psm	455.22
B	B	H2O	Mock	0.71
B	B	H2O	Mock	1.26
B	B	H2O	Mock	1.03
B	B	H2O	Psm	38.71
B	B	H2O	Psm	47.94
B	B	H2O	Psm	52.67
B	B	Pip	Mock	9.73
B	B	Pip	Mock	7.24
B	B	Pip	Mock	11.45
B	B	Pip	Psm	363.50
B	B	Pip	Psm	234.22
B	B	Pip	Psm	211.57
C	C	H2O	Mock	1.07
C	C	H2O	Mock	1.08
C	C	H2O	Mock	0.86
C	C	H2O	Psm	43.16
C	C	H2O	Psm	54.51
C	C	H2O	Psm	62.09
C	C	Pip	Mock	15.13
C	C	Pip	Mock	11.08
C	C	Pip	Mock	21.40
C	C	Pip	Psm	596.04
C	C	Pip	Psm	753.22
C	C	Pip	Psm	458.27

ANOVA results	SumSq	Df	F value	Pr(>F)
Treatment1	342691	1	133.2838	2.77*10 ⁻¹¹
Treatment2	557374	1	216.7811	1.63*10 ⁻¹³
Experiment	47633	2	9.2631	0.001044
Treatment1:Treatment2	304055	1	118.2569	9.33*10 ⁻¹¹
Treatment1:Experiment	41682	2	8.1057	0.002043
Treatment2:Experiment	44528	2	8.6592	0.001475
Treatment1:Treatment2:Experiment	38511	2	7.4891	0.002969
Residuals	61707	24		

Supplemental Table 2. Linear model-based analysis of the pipecolic acid-induced priming response in Col-0 plants to estimate treatment and experimental effect terms.

(A) Phenotype: relative *FMO1* transcript levels.

An analysis of variance (ANOVA) was performed with the depicted data (“object1”) from three independent experiments (A, B, and C), each consisting of three biological replicate samples per treatment type, using the R statistical package and the command:

“Anova(lm(Phenotype~Treatment1+Treatment2+Experiment+Treatment1*Treatment2+Treatment1*Experiment+Treatment2*Experiment+Treatment1*Treatment2*Experiment, data=object1),type=2)”.

Pip-induced priming was assessed as described in Fig. 8.

Treatment1: effect term for pre-treatment of plants (H₂O or Pip); Treatment2: effect term for the subsequent treatment of leaves (Mock or *Psm*).

Treatment1*Treatment2: effect term for the interaction of treatments (“Priming”).

Experiment: Term reflecting variation between experiments; Experiment*Treatmentx: Term reflecting experimental impact on effect of treatment x; Experiment*Treatment1*Treatment2: Term reflecting experimental influence on treatment interaction.

SumSq: type II-sum of squares. Df: degrees of freedom. Pr(>F): P-value associated with a corresponding F value.

(B)	Experiment	Treatment1	Treatment2	Phenotype			
A	A	H2O	Mock	0.45			
A	A	H2O	Mock	1.54			
A	A	H2O	Mock	1.01			
A	A	H2O	Psm	21.42			
A	A	H2O	Psm	16.66			
A	A	H2O	Psm	23.67			
A	A	Pip	Mock	11.00			
A	A	Pip	Mock	13.53			
A	A	Pip	Mock	20.39			
A	A	Pip	Psm	111.24			
A	A	Pip	Psm	92.73			
A	A	Pip	Psm	125.75			
B	B	H2O	Mock	1.16			
B	B	H2O	Mock	0.92			
B	B	H2O	Mock	0.92			
B	B	H2O	Psm	15.93			
B	B	H2O	Psm	14.89			
B	B	H2O	Psm	16.44			
B	B	Pip	Mock	3.58			
B	B	Pip	Mock	2.13			
B	B	Pip	Mock	5.75			
B	B	Pip	Psm	50.52			
B	B	Pip	Psm	41.10			
B	B	Pip	Psm	42.96			
C	C	H2O	Mock	1.17			
C	C	H2O	Mock	1.03			
C	C	H2O	Mock	0.80			
C	C	H2O	Psm	7.78			
C	C	H2O	Psm	4.98			
C	C	H2O	Psm	12.07			
C	C	Pip	Mock	10.61			
C	C	Pip	Mock	19.24			
C	C	Pip	Mock	17.40			
C	C	Pip	Psm	107.43			
C	C	Pip	Psm	114.60			
C	C	Pip	Psm	97.17			
ANOVA results				SumSq	Df	F value	Pr(>F)
Treatment1				15388	1	410.643	$< 2.2 \times 10^{-16}$
Treatment2				17987.7	1	480.019	$< 2.2 \times 10^{-16}$
Experiment				2786.8	2	37.184	4.45×10^{-08}
Treatment1:Treatment2				8557.2	1	228.356	9.24×10^{-14}
Treatment1:Experiment				2934.4	2	39.154	2.78×10^{-08}
Treatment2:Experiment				1374.7	2	18.343	1.46×10^{-05}
Treatment1:Treatment2:Experiment				1432.6	2	19.115	1.08×10^{-05}
Residuals				899.4	24		

Supplemental Table 2. (B) Phenotype: relative *ALD1* transcript levels.

(C)	Experiment	Treatment1	Treatment2	Phenotype			
A		H2O	Mock	0.61			
A		H2O	Mock	0.86			
A		H2O	Mock	1.53			
A		H2O	Psm	6.52			
A		H2O	Psm	8.88			
A		H2O	Psm	7.21			
A		Pip	Mock	13.97			
A		Pip	Mock	9.15			
A		Pip	Mock	14.91			
A		Pip	Psm	51.64			
A		Pip	Psm	27.18			
A		Pip	Psm	35.32			
B		H2O	Mock	0.74			
B		H2O	Mock	0.85			
B		H2O	Mock	1.41			
B		H2O	Psm	3.16			
B		H2O	Psm	3.74			
B		H2O	Psm	3.19			
B		Pip	Mock	3.79			
B		Pip	Mock	1.93			
B		Pip	Mock	4.41			
B		Pip	Psm	21.52			
B		Pip	Psm	12.75			
B		Pip	Psm	12.27			
C		H2O	Mock	0.62			
C		H2O	Mock	1.40			
C		H2O	Mock	0.98			
C		H2O	Psm	12.03			
C		H2O	Psm	6.48			
C		H2O	Psm	9.83			
C		Pip	Mock	5.03			
C		Pip	Mock	2.64			
C		Pip	Mock	2.85			
C		Pip	Psm	29.78			
C		Pip	Psm	36.85			
C		Pip	Psm	64.38			
ANOVA results				SumSq	Df	F value	Pr(>F)
Treatment1				2182.91	1	48.5717	3.31*10 ⁻⁰⁷
Treatment2				2257.04	1	50.2212	2.52*10 ⁻⁰⁷
Experiment				620.11	2	6.899	0.0042944
Treatment1:Treatment2				909.73	1	20.2422	0.0001484
Treatment1:Experiment				321.96	2	3.5819	0.0435227
Treatment2:Experiment				436.37	2	4.8548	0.0169623
Treatment1:Treatment2:Experiment				182.39	2	2.0292	0.1533831
Residuals				1078.61	24		

Supplemental Table 2. (C) Phenotype: relative *PR1* transcript levels.

(D)	Experiment	Treatment1	Treatment2	Phenotype			
A	A	H2O	Mock	0.07			
A	A	H2O	Mock	0.23			
A	A	H2O	Mock	0.14			
A	A	H2O	Psm	0.78			
A	A	H2O	Psm	0.63			
A	A	H2O	Psm	1.09			
A	A	Pip	Mock	0.24			
A	A	Pip	Mock	0.27			
A	A	Pip	Mock	0.13			
A	A	Pip	Psm	4.44			
A	A	Pip	Psm	4.16			
A	A	Pip	Psm	3.88			
B	B	H2O	Mock	0.01			
B	B	H2O	Mock	0.12			
B	B	H2O	Mock	0.02			
B	B	H2O	Psm	0.19			
B	B	H2O	Psm	0.39			
B	B	H2O	Psm	0.45			
B	B	Pip	Mock	0.23			
B	B	Pip	Mock	0.52			
B	B	Pip	Mock	0.10			
B	B	Pip	Psm	3.05			
B	B	Pip	Psm	5.31			
B	B	Pip	Psm	4.93			
C	C	H2O	Mock	0.30			
C	C	H2O	Mock	0.23			
C	C	H2O	Mock	0.13			
C	C	H2O	Psm	1.05			
C	C	H2O	Psm	0.58			
C	C	H2O	Psm	0.85			
C	C	Pip	Mock	0.49			
C	C	Pip	Mock	0.73			
C	C	Pip	Mock	0.66			
C	C	Pip	Psm	10.96			
C	C	Pip	Psm	8.78			
C	C	Pip	Psm	14.80			
ANOVA results				SumSq	Df	F value	Pr(>F)
Treatment1				88.172	1	95.713	7.53*10 ⁻¹⁰
Treatment2				105.473	1	114.493	1.29*10 ⁻¹⁰
Experiment				31.89	2	17.308	2.22*10 ⁻⁰⁵
Treatment1:Treatment2				75.864	1	82.352	3.16*10 ⁻⁰⁹
Treatment1:Experiment				26.314	2	14.282	8.21*10 ⁻⁰⁵
Treatment2:Experiment				24.026	2	13.041	0.0001467
Treatment1:Treatment2:Experiment				22.913	2	12.436	0.0001967
Residuals				22.109	24		

Supplemental Table 2. (D) Phenotype: Camalexin levels ($\mu\text{g g}^{-1}$ FW).

Experiment	Treatment1	Treatment2	Phenotype
A	H2O	Mock	1.5
A	H2O	Mock	0.2
A	H2O	Mock	1.3
A	H2O	SA	585.1
A	H2O	SA	336.0
A	H2O	SA	625.5
A	Pip	Mock	111.5
A	Pip	Mock	114.4
A	Pip	Mock	103.7
A	Pip	SA	1270.1
A	Pip	SA	1990.2
A	Pip	SA	1291.4
B	H2O	Mock	0.9
B	H2O	Mock	1.4
B	H2O	Mock	0.7
B	H2O	SA	2260.9
B	H2O	SA	2165.2
B	H2O	SA	2253.5
B	Pip	Mock	1443.6
B	Pip	Mock	803.6
B	Pip	Mock	1377.9
B	Pip	SA	8419.1
B	Pip	SA	7856.1
B	Pip	SA	15973.9
C	H2O	Mock	0.7
C	H2O	Mock	0.6
C	H2O	Mock	1.7
C	H2O	SA	227.9
C	H2O	SA	497.1
C	H2O	SA	669.6
C	Pip	Mock	51.5
C	Pip	Mock	180.6
C	Pip	Mock	47.0
C	Pip	SA	3071.4
C	Pip	SA	1121.0
C	Pip	SA	1765.3

ANOVA results	SumSq	Df	F value	Pr(>F)
Treatment1	38776567	1	21.2372	0.0001124
Treatment2	64364518	1	35.2512	3.98*10 ⁻⁰⁶
Experiment	70169530	2	19.2153	1.04*10 ⁻⁰⁵
Treatment1:Treatment2	23220994	1	12.7177	0.0015636
Treatment1:Experiment	35112399	2	9.6152	0.0008572
Treatment2:Experiment	46415765	2	12.7105	0.000172
Treatment1:Treatment2:Experiment	19049097	2	5.2164	0.0131481
Residuals	43821113	24		

Supplemental Table 3. Linear model-based analysis of the amplification of salicylic acid-induced *PR1* expression by pipercolic acid in Col-0 plants to estimate treatment and experimental effect terms. Phenotype: relative *PR1* transcript levels.

An analysis of variance (ANOVA) was performed with the depicted data ("object1") from three independent experiments (A, B, and C), each consisting of three biological replicate samples per treatment type, using the R statistical package and the command:

"Anova(lm(Phenotype~Treatment1+Treatment2+Experiment+Treatment1*Treatment2+Treatment1*Experiment+Treatment2*Experiment+Treatment1*Treatment2*Experiment, data=object1),type=2)".

Pip- and SA-treatments were performed as described in Fig. 9.

Treatment1: effect term for pre-treatment of plants (H₂O or Pip); Treatment2: effect term for the subsequent treatment of leaves (Mock or SA).

Treatment1*Treatment2: effect term for the interaction of treatments ("Priming").

Experiment: Term reflecting variation between experiments; Experiment*Treatmentx: Term reflecting experimental impact on effect of treatment x; Experiment*Treatment1*Treatment2: Term reflecting experimental influence on treatment interaction.

SumSq: type II-sum of squares. Df: degrees of freedom. Pr(>F): P-value associated with a corresponding F value.

Primer name	Primer sequence (5' to 3')	Usage
<i>ald1-fw</i>	TTACGATGCATTTGCTATGACC	Left primer; genotyping of <i>sid2-1 ald1</i>
<i>ald1-rv</i>	TTTTAAATGGAACGCAAGGAG	Right primer; genotyping of <i>sid2-1 ald1</i>
<i>ICS1-FW</i>	GTATATGTGACAGAGTTGTTGTC	Sequencing primer
LB	TGGTTCACGTAAGTGGCCATC	T-DNA Left Border primer
<i>ALD1-FW</i>	GTGCAAGATCCTACCTTCCCGGC	qRT-PCR
<i>ALD1-RV</i>	CGGTCCTTGGGGTCATAGCCAGA	qRT-PCR
<i>ARD3-FW</i>	CATGGACTTATGTGAGGTGTG	qRT-PCR
<i>ARD3-RV</i>	ACATCAAAGTATCCACTTCCTG	qRT-PCR
<i>FMO1-FW</i>	TCTTCTGCGTGCCGTAGTTTC	qRT-PCR
<i>FMO1-RV</i>	CGCCATTTGACAAGAAGCATAG	qRT-PCR
<i>ICS1-FV</i>	GCAAGAGTGCAACATCTATATTCTC	qRT-PCR; genotyping of <i>sid2-1 ald1</i>
<i>ICS1-RV</i>	CACAAACAGCTGGAGTTGGA	qRT-PCR; genotyping of <i>sid2-1 ald1</i>
<i>PR-1-FW</i>	GTGCTCTTGTCTTCCCTCG	qRT-PCR
<i>PR-1-RV</i>	GCCTGGTTGTGAACCCTTAG	qRT-PCR
<i>SAG13-FV</i>	GCGACAACATAAGGACGA	qRT-PCR
<i>SAG13-RV</i>	CTTCATTTGCTTCTCCAACAC	qRT-PCR
<i>GRXS13-FV</i>	GGTTGAGATTGGTGAAGAAGAC	qRT-PCR
<i>GRXS13-RV</i>	GCCATTAATATGAGCAGCCA	qRT-PCR
<i>PTB-FW</i>	GATCTGAATGTTAAGGCTTTTAGCG	qRT-PCR; reference gene
<i>PTB-RV</i>	GGCTTAGATCAGGAAGTGTATAGTCTCTG	qRT-PCR; reference gene

Supplemental Table 4. List of primers used in this study.