New Phytologist **Supporting Information**

Article title: Insect eggs trigger systemic acquired resistance against a fungal and an oomycete pathogen

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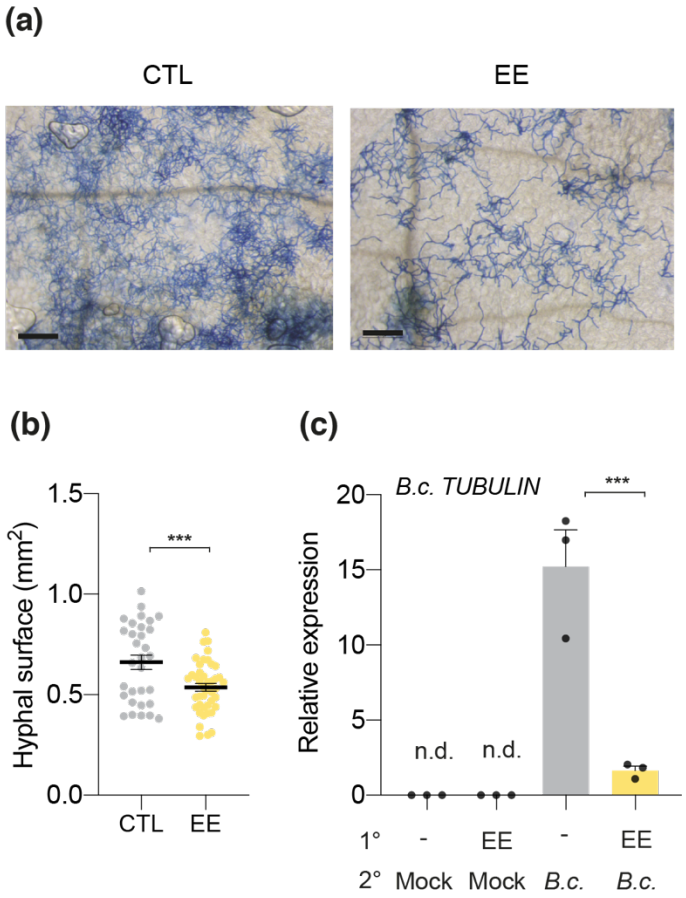

Fig. S1 Egg extract treatment reduces *Botrytis cinerea* growth in distal leaves. (a) Photographs of stained hyphae on control plants (top) and plants pretreated with *Pieris brassicae* egg extract (EE) (bottom, distal leaf), 2 days post-inoculation. Scale bar: 200 µm. (b) Plants were pretreated with EE and hyphal growth was measured 2 days after inoculation. Hyphae were stained by trypan blue and the surface of hyphae was quantified with ImageJ. Means ± SE of three independent experiments are shown (n = 8-14 per experiment). Significant differences between control and treated plants are indicated (linear mixed model, *** P<0.001. (c) Expression of the *B. cinerea* tubulin gene in distal leaves. Local leaves (1°) were either treated with EE for 5 days or not treated (-). Distal leaves (2°) were then inoculated with PDB (Mock) or *B. cinerea* spore suspension (*B.c.*) for 2 days. Means ± SE of three independent experiments are shown (n = 10-12 per experiment). Significant differences between control and treated plants are indicated (linear mixed model, *** P<0.001. n.d. not determined.

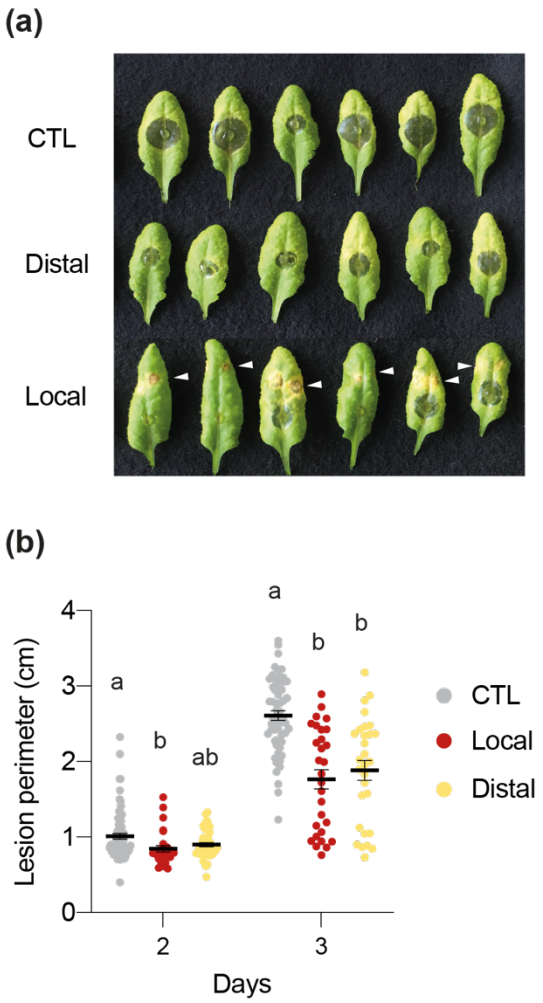

Fig. S2 Time course of egg extract-induced reduction of *Botrytis cinerea* infection. (a) A solution of *B. cinerea* spores was deposited on untreated plants (CTL), on leaves distal to *Pieris brassicae* egg extract (EE)-treated leaves, or on EE-treated leaves. White arrows indicate the application site of the EE. Photographs were taken 3 days after infection. (b) Lesion perimeter measurement of control leaves, EE-treated leaves and leaves distal from EE-treated plants. Means ± SE of three independent experiments are shown (n = 8-37 per experiment). For each time point, different letters indicate significant differences at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test). Dots indicate individual values.

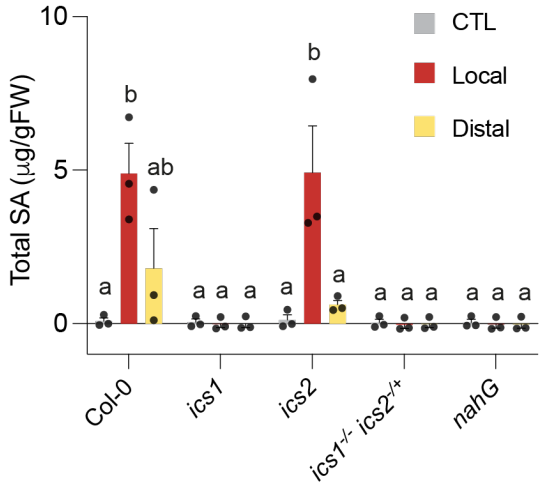

Fig. S3 Salicylic acid (SA) quantification in SA biosynthesis mutants. Total SA was measured in untreated plants (CTL), *Pieris brassicae* egg extract (EE)-treated leaves (Local) and in leaves distal to EE-treated leaves (Distal) after 5 days. Means ± SE of three independent experiments are shown (n = 6 per experiment). Different letters indicate significant difference between treatments within genotypes at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test).

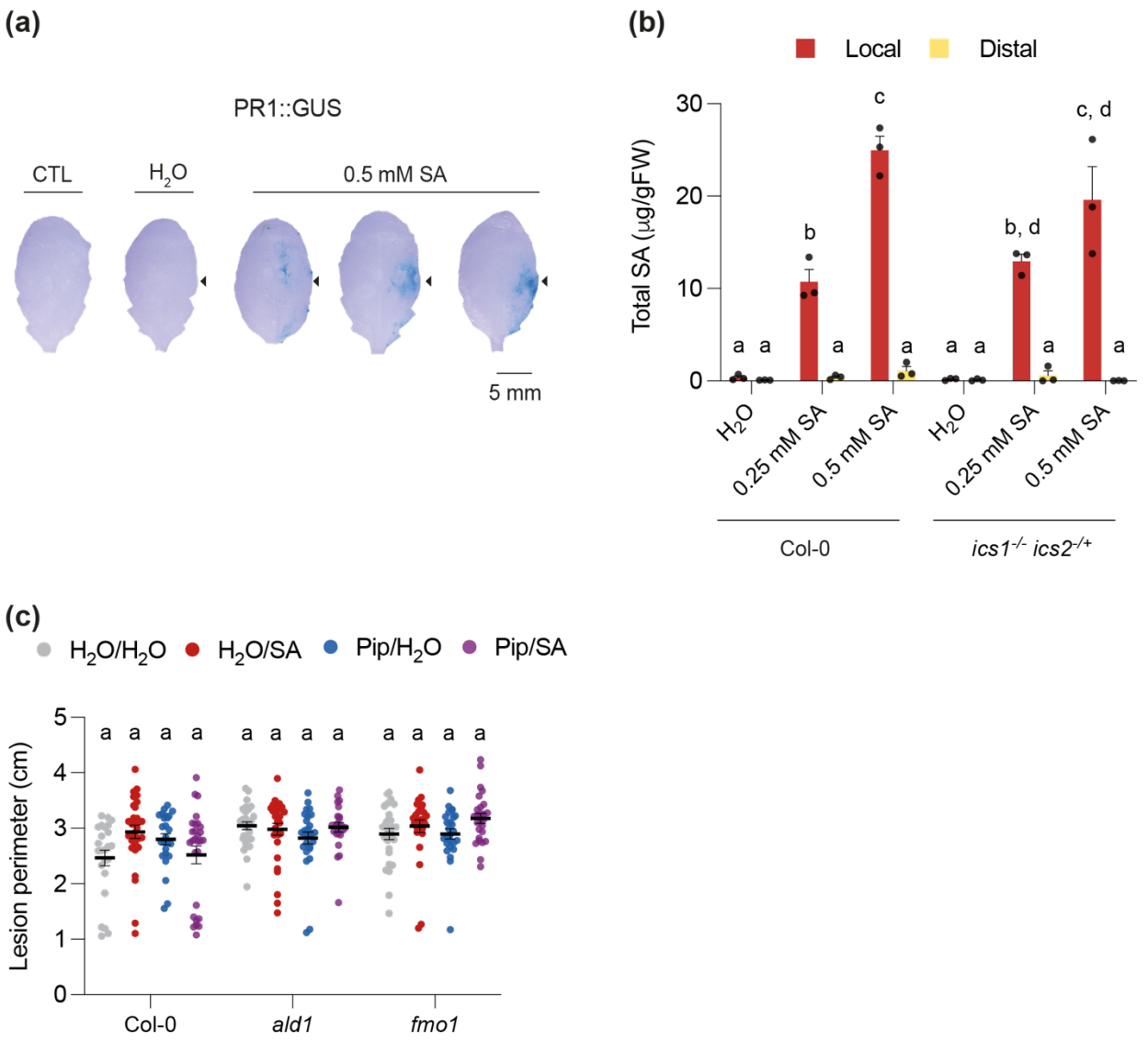

Fig. S4 Exogenous salicylic acid (SA) infiltration does not trigger egg extract-induced systemic acquired ressitance. (a). Infiltration of H_2O and 0.5 mM SA in PR1::GUS reporter line. Black triangles indicate which half of the leaf was infiltrated. For SA infiltration, three representative images from different plants are shown. CTL, untreated. (b). Plant genotypes were infiltrated with H_2O, 0.25 mM and 0.5 mM of SA in the abaxial surface of two leaves per plant for 4 h before SA quantification in local (infiltrated leaves) and distal leaves. Means ± SE of three independent experiments are shown (n = 6 per experiment). The double mutant *ics1 ics2* was homozygous for *ics1* ($^{-/-}$) and heterozygous for *ics2* ($^{-/+}$). For each genotype and location, different letters indicate significant differences between treatments in local leaves at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test) (c). H_2O or 1 mM Pip solution was applied to the soil 24 h prior infection with *Botrytis cinerea* for 3 days. H_2O or 0.25 mM SA were infiltrated in two leaves per plant 4 h prior infection. Means ± SE of three independent experiments are shown (n = 6-12 per experiment). For each genotype, different letters indicate significant differences at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test). Dots indicate individual values.

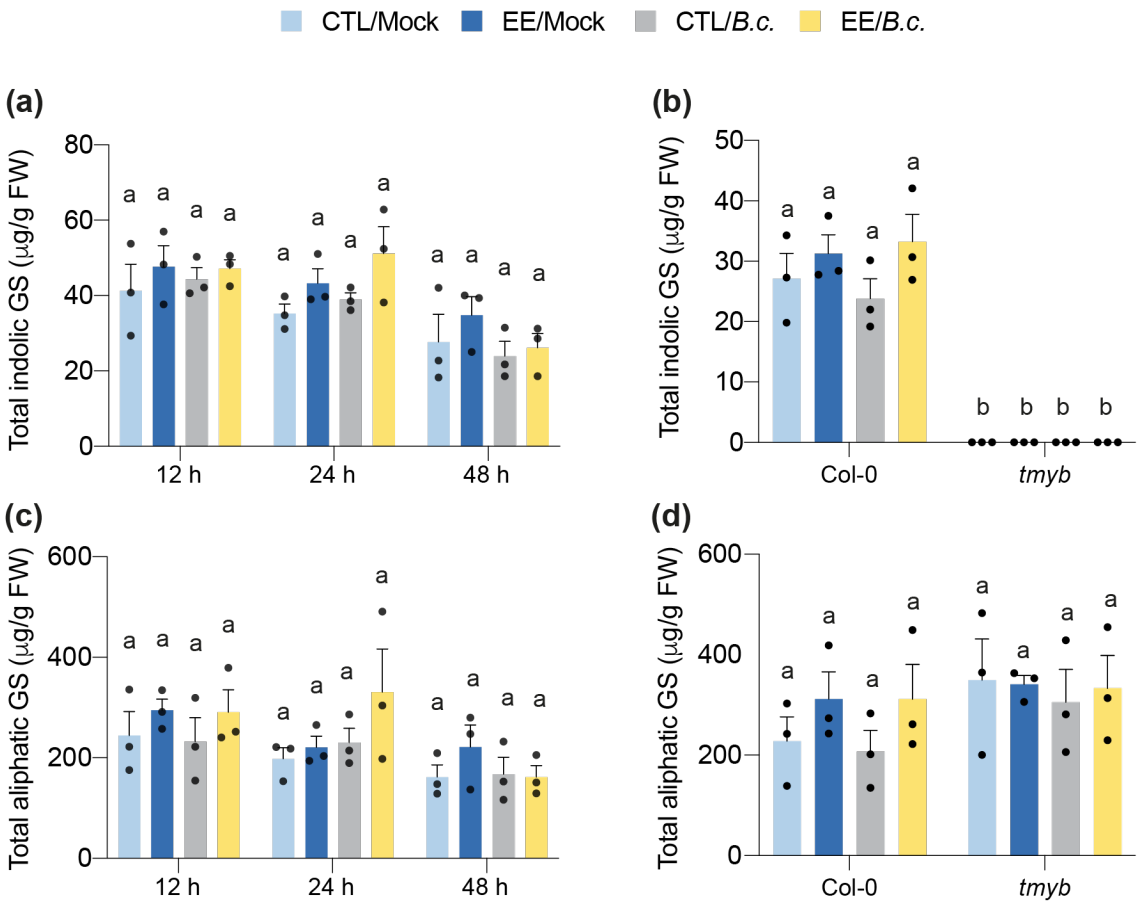

Fig. S5 Glucosinolates levels in egg extract- and *Botrytis cinerea*-treated plants. Levels of indolic glucosinolates (a,b) and aliphatic glucosinolates (c,d)) were quantified in distal leaves from 12 h to 48 h after *B. cinerea* infection in Col-0 (a,c) or after 24 h in Col-0 and *tmyb* (b,d). Local leaves were pretreated with *Pieris brassicae* egg extract (EE) for 5 days or left untreated (CTL) and then distal leaves were infected with *B. cinerea* (*B.c.*) or a mock solution (Mock). Means ± SE of three independent experiments are shown (n = 10-12 per experiment). For each time point (a,c) or between genotypes (b,d), different letters indicate significant differences at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test). *tmyb* = *myb34 myb51 myb122*.

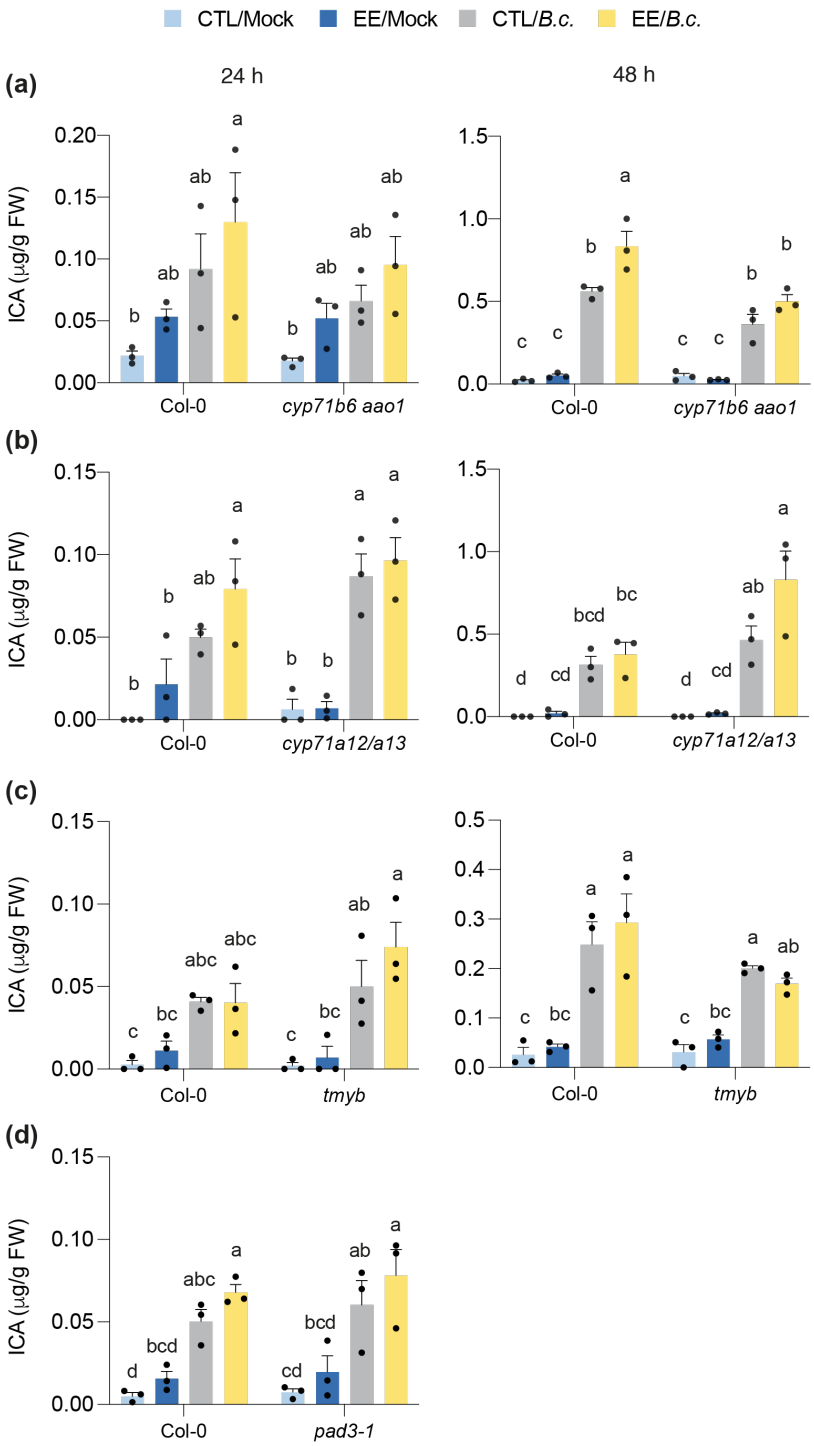

Fig. S6 Indole carboxylic acid (ICA) accumulates in response to *Botrytis cinerea* infection. (a-d) Levels of ICA were quantified in distal leaves from 24 h and 48 h after *B. cinerea* infection. Local leaves were pretreated with *Pieris brassicae* egg extract (EE) for 5 days or left untreated (CTL) and then distal leaves were infected with *B. cinerea* (*B.c.*) or a mock solution (Mock). Means ± SE of three independent experiments are shown (n = 10-12 per experiment). Different letters indicate significant differences at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test). *tmyb* = *myb34 myb51 myb122*.

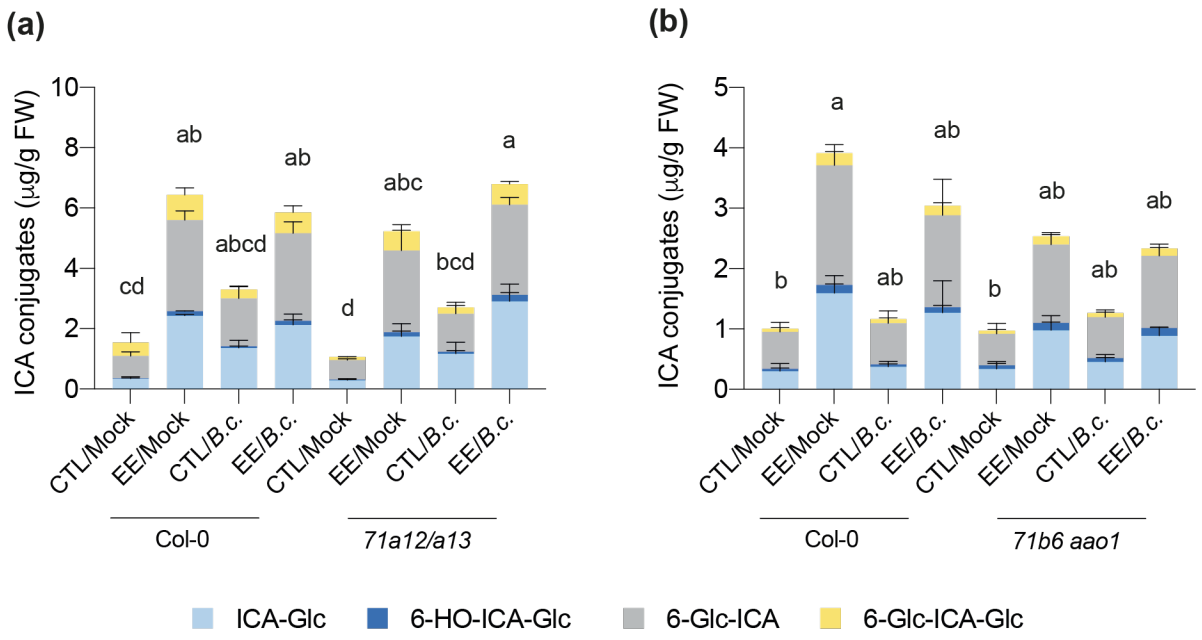

Fig. S7 Indole carboxylic acid (ICA) conjugates accumulate in response to egg extract treatment. (a,b) Levels of ICA conjugates were quantified in distal leaves 48 h after *Botrytis cinerea* infection. Local leaves were pretreated with *Pieris brassicae* egg extract (EE) for 5 days or left untreated (CTL) and then distal leaves were infected with *B. cinerea* (*B.c.*) or a mock solution (Mock). Means ± SE of three independent experiments are shown (n = 10-12 per experiment). Different letters indicate significant differences at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test).

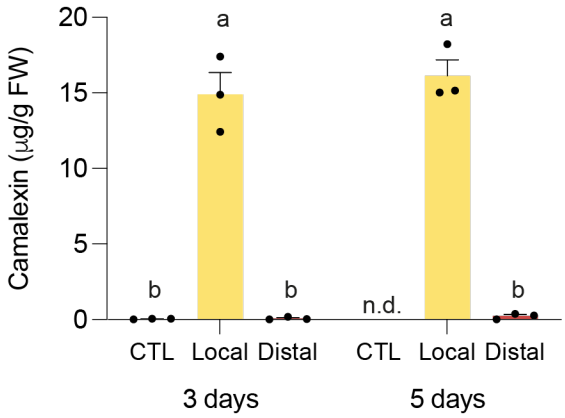

Fig. S8 Egg extract induces camalexin accumulation in local leaves. Levels of camalexin were quantified in local and distal leaves 3 and 5 days after *Pieris brassicae* egg extract treatment. Control (CTL) leaves were untreated. Means ± SE of three independent experiments are shown (n = 10-12 per experiment). Different letters indicate significant differences at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test). n.d., not determined.

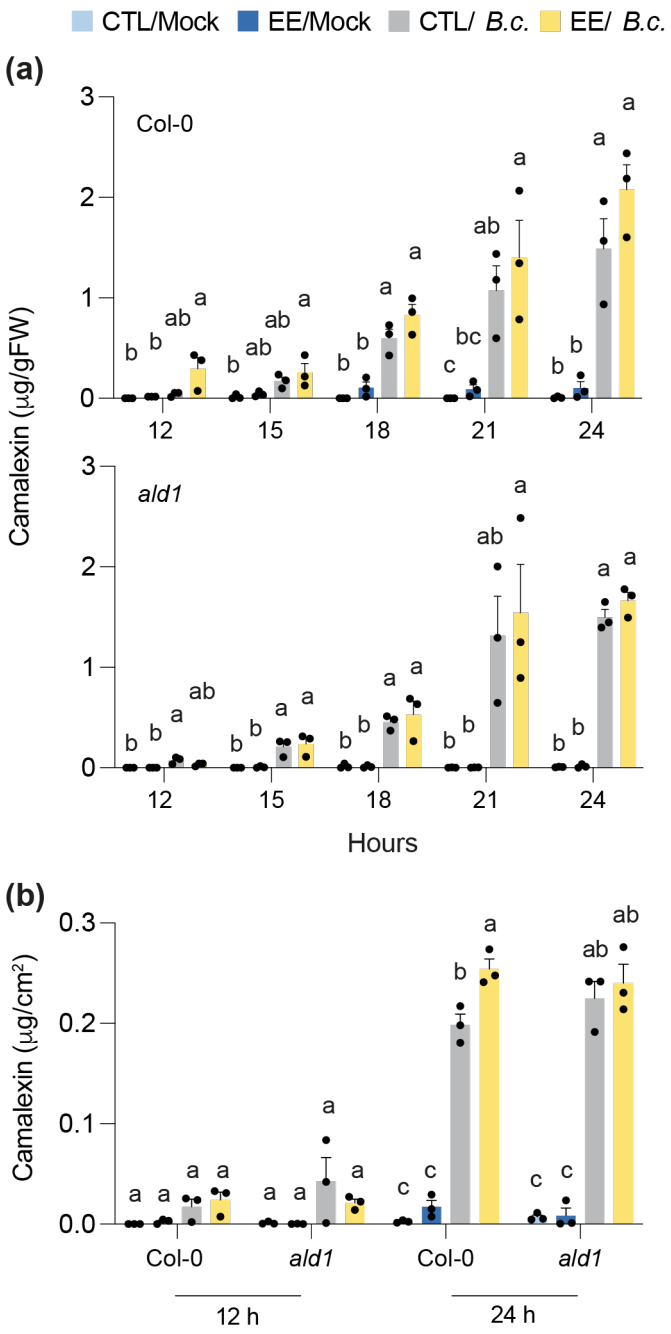

Fig. S9 Early time course of camalexin accumulation. Levels of camalexin were quantified in distal leaves from 12 h to 24 h after *Botrytis cinerea* infection. Local leaves were pretreated with *Pieris brassicae* egg extract (EE) for 5 days or left untreated (CTL) and then distal leaves were infected with *B. cinerea* (*B.c.*) or a mock solution (Mock). (a) Total and (b) leaf surface camalexin was analyzed. Means ± SE of three independent experiments are shown (n = 8-12 per experiment). For each time point, different letters indicate significant differences at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test).

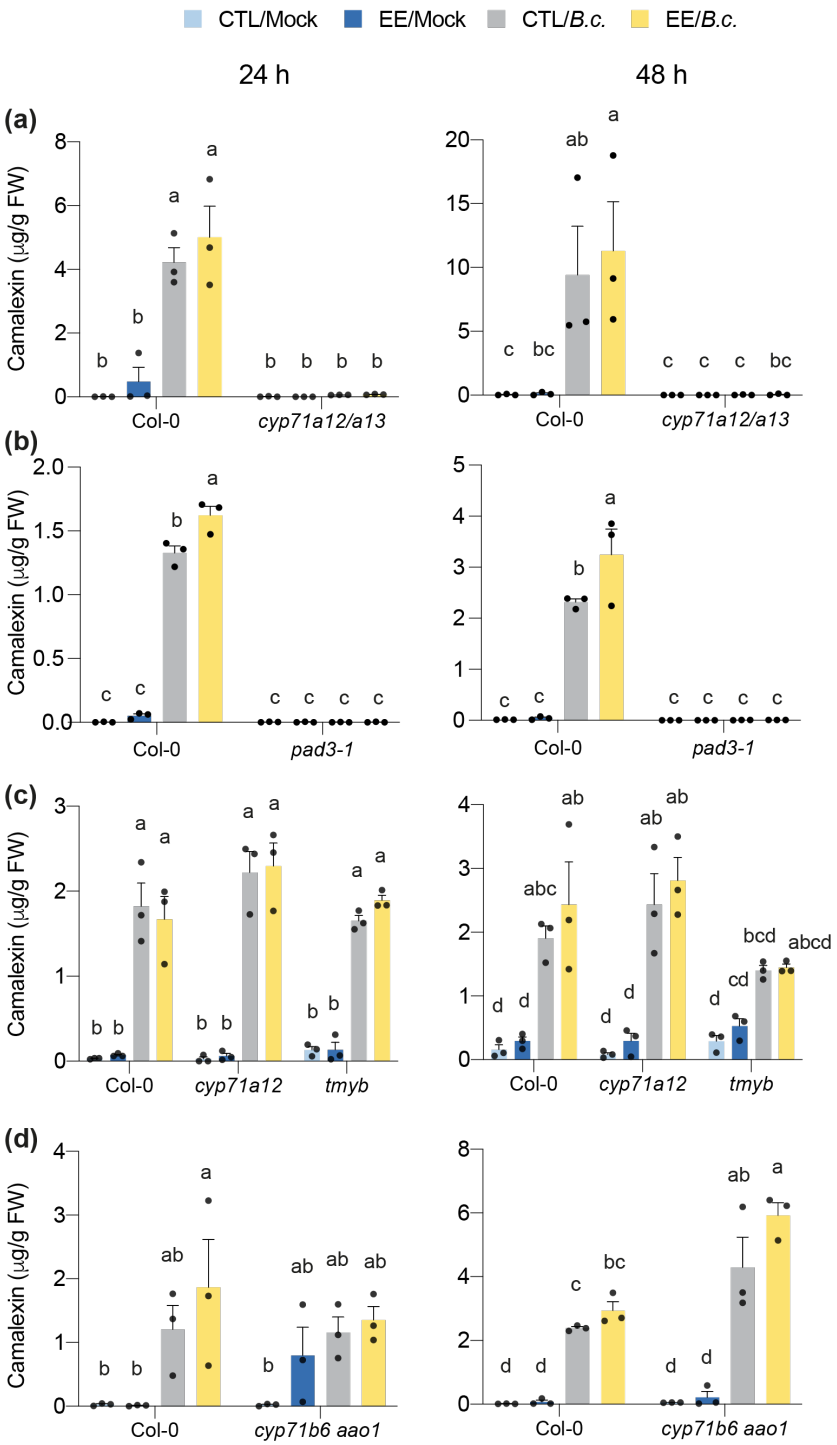

Fig. S10 Camalexin levels in various indolic mutants. (a-d) Levels of camalexin were quantified in distal leaves from 24 h and 48 h after *Botrytis cinerea* infection. Local leaves were pretreated with *Pieris brassicae* egg extract (EE) for 5 days or left untreated (CTL) and then distal leaves were infected with *B. cinerea* (*B.c.*) or a mock solution (Mock). Means ± SE of three independent experiments are shown (n = 10-12 per experiment). Different letters indicate significant differences at $P<0.05$ (ANOVA followed by Tukey's Honest Significant Difference test). *tmyb* = *myb34 myb51 myb122*.

Table S2 Time-course of single glucosinolate species accumulation.

Metabolite	Abb.	Col-0 12 h							
		CTL/Mock		EE/Mock		CTL/*B.c.*		EE/*B.c.*	
Glucoiberin[1]	3MSOP	20.55	± 4.33	25.25	± 2.00	21.22	± 3.20	26.41	± 2.63
Glucoraphanin[1]	4MSOB	141.15	± 33.5	173.7	± 16.9	135.29	± 30.2	175.69	± 30.6
Glucoalyssin[1]	5MSOP	4.76	± 0.99	5.65	± 0.64	4.29	± 0.83	5.8	± 0.95
Glucoibarin[1]	7MSOH	2.8	± 0.77	3.18	± 0.38	2.23	± 0.33	3.3	± 0.57
Glucoerucin[1]	4MTB	75.3	± 8.89	86.91	± 2.98	69.15	± 13.3	79.34	± 10.1
Glucobrassicin[2]	I3M	32.05	± 5.97	35.93	± 4.73	34.47	± 2.37	35.26	± 1.64
Hydroxyglucobrassicin[2]	OH-I3M	3.95	± 0.64	4.47	± 0.52	4.24	± 0.32	4.53	± 0.17
Methoxyglucobrassicin[2]	4MOI3M	4.75	± 0.44	6.57	± 0.42	5.13	± 0.35	6.7	± 0.61
Neoglucobrassicin[2]	1MOI3M	0.57	± 0.06	0.69	± 0.05	0.52	± 0.05	0.66	± 0.04
		Col-0 24 h							
		CTL/Mock		EE/Mock		CTL/*B.c.*		EE/*B.c.*	
Glucoiberin[1]	3MSOP	17.81	± 2.38	23.29	± 3.04	21.41	± 3.47	28.38	± 7.21
Glucoraphanin[1]	4MSOB	121.09	± 13.3	126.85	± 14.1	136.67	± 19.6	199.84	± 58.6
Glucoalyssin[1]	5MSOP	4.12	± 0.41	4.99	± 0.66	4.58	± 0.28	7.04	± 1.39
Glucoibarin[1]	7MSOH	2.41	± 0.25	2.77	± 0.23	2.59	± 0.19	4.34	± 0.75
Glucoerucin[1]	4MTB	52.42	± 6.00	63.03	± 11.6	64.97	± 5.63	91.19	± 20.6
Glucobrassicin[2]	I3M	26.02	± 1.47	32.05	± 2.81	28.5	± 1.31	36.58	± 5.25
Hydroxyglucobrassicin[2]	OH-I3M	3.23	± 0.26	4	± 0.40	3.57	± 0.16	4.82	± 0.74
Methoxyglucobrassicin[2]	4MOI3M	5.45	± 0.73	6.61	± 0.56	6.29	± 0.33	8.95	± 1.06
Neoglucobrassicin[2]	1MOI3M	0.53	± 0.10	0.61	± 0.13	0.6	± 0.02	0.79	± 0.13
		Col-0 48 h							
		CTL/Mock		EE/Mock		CTL/*B.c.*		EE/*B.c.*	
Glucoiberin[1]	3MSOP	15.16	± 2.25	19.87	± 3.31	14.93	± 2.86	15.09	± 1.42
Glucoraphanin[1]	4MSOB	92.95	± 15.1	126.81	± 23.9	90.99	± 14.5	90.08	± 9.98
Glucoalyssin[1]	5MSOP	3.42	± 0.62	4.49	± 0.60	3.32	± 0.59	3.46	± 0.32
Glucoibarin[1]	7MSOH	2.2	± 0.33	2.77	± 0.50	1.81	± 0.31	2.05	± 0.25
Glucoerucin[1]	4MTB	48.18	± 6.95	67.64	± 15.9	56.15	± 16.5	51.3	± 10.9
Glucobrassicin[2]	I3M	19.18	± 5.96	24.28	± 3.94	14.82	± 2.65	15.71	± 3.02
Hydroxyglucobrassicin[2]	OH-I3M	2.35	± 0.68	3.13	± 0.49	1.92	± 0.35	2.13	± 0.39
Methoxyglucobrassicin[2]	4MOI3M	5.64	± 0.63	6.76	± 0.49	6.33	± 0.86	7.1	± 0.30
Neoglucobrassicin[2]	1MOI3M	0.53	± 0.13	0.66	± 0.13	0.86	± 0.10	1.18	± 0.34

Levels of single aliphatic[1]and indole[2] glucosinolate species in µg/g FW, quantified in distal leaves from 12 h to 48 h after *Botrytis cinerea* (*B.c.*) infection or treatment with a mock solution (Mock) in Col-0, with or without pretreatment for 5 days with *Pieris brassicae* egg extract (EE). Means ± SE of three independent experiments are shown (n = 10-12 leaves per sample/experiment). Total aliphatic and indole glucosinolates are shown in Fig. S5a and c. Abb. = Abbreviation.

Table S3 Single glucosinolate species in Col-0 and *tmyb* mutant.

Metabolite	Abb.	Col-0							
		CTL/Mock		EE/Mock		CTL/*B.c.*		EE/*B.c.*	
Glucoiberin[1]	3MSOP	16.97	± 3.59	22.28	± 3.07	16.25	± 3.30	22.93	± 4.42
Glucoraphanin[1]	4MSOB	114.18	± 23.3	165.43	± 29.8	107.13	± 22.2	170.10	± 39.6
Glucoalyssin[1]	5MSOP	3.17	± 0.63	4.04	± 0.54	3.01	± 0.59	4.29	± 0.81
Glucoibarin[1]	7MSOH	1.32	± 0.54	1.71	± 0.40	1.08	± 0.37	1.66	± 0.52
Glucohirsutin[1]	8MSOO	16.17	± 4.98	20.78	± 3.87	12.89	± 3.62	19.86	± 3.59
Glucoerucin[1]	4MTB	40.12	± 7.84	56.9	± 11.9	36.08	± 8.08	52.58	± 14.4
Glucoberteroin[1]	5MTB	3.47	± 0.64	4.29	± 0.57	3.11	± 0.66	4.31	± 1.02
Gluconasturtiin[1]	2PE	0.64	± 0.18	0.88	± 0.24	0.59	± 0.16	0.85	± 0.29
7-Methylthioheptyl-GS[1]	7MTH	7.19	± 1.73	7.72	± 1.07	6.48	± 1.19	7.74	± 1.84
8-Methylthiooctyl-GS[1]	8MTO	24.91	± 5.31	27.68	± 2.78	20.27	± 3.21	26.51	± 4.74
Glucobrassicin[2]	I3M	20.22	± 3.23	23.53	± 2.35	18.11	± 2.59	25.54	± 3.55
Hydroxyglucobrassicin[2]	OH-I3M	2.3	± 0.40	2.85	± 0.35	2.32	± 0.42	3.38	± 0.74
Methoxyglucobrassicin[2]	4MOI3M	4.62	± 0.58	4.88	± 0.61	3.37	± 0.27	4.3	± 0.33
		tmyb							
		CTL/Mock		EE/Mock		CTL/*B.c.*		EE/*B.c.*	
Glucoiberin[1]	3MSOP	25.40	± 4.21	25.64	± 1.9	23.01	± 3.80	24.47	± 4.54
Glucoraphanin[1]	4MSOB	169.51	± 40.4	170.85	± 6.9	150.64	± 31.8	158.51	± 32.5
Glucoalyssin[1]	5MSOP	4.58	± 0.55	4.42	± 0.6	4.13	± 0.62	4.38	± 0.81
Glucoibarin[1]	7MSOH	2.43	± 0.75	2.24	± 0.2	2.06	± 0.74	2.36	± 0.73
Glucohirsutin[1]	8MSOO	28.56	± 9.87	24.63	± 0.9	22.89	± 6.74	24.69	± 6.31
Glucoerucin[1]	4MTB	70.87	± 21.8	69.56	± 3.3	62.64	± 16.9	75.05	± 15.8
Glucoberteroin[1]	5MTB	4.92	± 0.66	5.08	± 0.4	4.3	± 0.57	4.86	± 0.78
Gluconasturtiin[1]	2PE	1.09	± 0.27	1.09	± 0.2	0.99	± 0.28	1.16	± 0.24
7-Methylthioheptyl-GS[1]	7MTH	10.91	± 1.12	9.94	± 1.7	9.37	± 1.34	10.2	± 2.18
8-Methylthiooctyl-GS[1]	8MTO	30.97	± 5.29	27.49	± 3.5	25.21	± 3.43	27.06	± 4.46
Glucobrassicin[2]	I3M	n.d.		n.d.		n.d.		n.d.	
Hydroxyglucobrassicin[2]	OH-I3M	n.d.		n.d.		n.d.		n.d.	
Methoxyglucobrassicin[2]	4MOI3M	n.d.		n.d.		n.d.		n.d.	

Levels of single aliphatic[1] and indole[2] glucosinolate species in µg/g FW, quantified in distal leaves 24 h after *Botrytis cinerea* infection or treatment with a mock solution (Mock) in Col-0 and *tmyb*, with or without pretreatment for 5 days with *Pieris brassicae* egg extrcat (EE). Means ± SE of three independent experiments are shown (n = 10-12 leaves per sample/experiment). Total aliphatic and indole glucosinolates are shown in Fig. S5b and d. Abb. = Abbreviation, n.d. = not detectable. *tmyb* = *myb34 myb51 myb122*.

Methods S1 Lines used in this study.

Lines used in this study: *ald1* (Návarová *et al.*, 2012), *cyp71a12* (Millet *et al.*, 2010), *cyp71a12 cyp71a13* (Müller *et al.*, 2015), *cyp79b2 cyp79b3* (Zhao *et al.*, 2002), *cyp82c2-2* (Rajniak *et al.*, 2015), *fmo1* (Mishina & Zeier, 2006), *fox1* (Rajniak *et al.*, 2015), *ics1* (Nawrath & Métraux, 1999), *ics2* (Garcion *et al.*, 2008), *lecrk-I.8* (Gouhier-Darimont *et al.*, 2013), *myb28 myb29* (Beekwilder *et al.*, 2008), *myb34 myb51 myb122* (*tmyb*) (Frerigmann & Gigolashvili, 2014), *nahG* (Nawrath & Métraux, 1999), *npr1-1* (Cao *et al.*, 1997), *npr1-1 npr4-4D* (Liu *et al.*, 2020), *pad3-1* (Glazebrook & Ausubel, 1994), *wrky33* (Birkenbihl *et al.*, 2012). All genotypes were in the Columbia (Col-0) background. The *cyp71b6 aao1* double mutant was obtained by crossing single mutants described previously, *cyp71b6* (GABI_305A04) and *aao1* (SALK_069221) (Müller *et al.*, 2019). Genotyping was done using the following primers: CYP71B6 (At2g24180) LP: 5'-CCAGGTGCTTCTTCAACACTC-3', RP: 5'-TCATCTGGATCTTCCGTTGAC-3'; AAO1 (At5g20960) LP: 5'-AGCAGCTCGAGTCAAGAACAG-3', RP: 5'-TGCAATATCTGCATGCTTTTG-3'. The *ics1*$^{-/-}$ *ics2*$^{+/-}$ double mutant (homozygous for *ics1*, heterozygous for *ics2*) was obtained by crossing *ics1* and *ics2*, and was genotyped using a CAPS marker for *ics1* (Heck *et al.*, 2003) and flanking primers for *ics2* T-DNA knockout (Garcion *et al.*, 2008). *ICS1* (At1g74710) Fw: 5'-GGA CTC AAT TAG GTG TCT GC-3', Rv: 5'-AAG CCT TGC TTC TTC TGC TG-3'; *ICS2* (At1g18870) Fw: 5'-GTC TTC AAA GTC TCC TCT GAT-3'Rv: 5'-TGA ATC ACC TCT AGG CCT TGT-3'.

Methods S2 Measurement of *Botrytis cinerea* growth by QPCR.

Total RNA from a pool of 10-12 leaves was extracted using a ReliaPrepTM RNA Tissue Miniprep System (Promega). For cDNA synthesis, 500 ng of total RNA was reverse-transcribed using M-MLV reverse transcriptase (Invitrogen) in a final volume of 15.25 µl. Each cDNA sample was generated in triplicate and diluted eightfold with water. Quantitative real-time PCR analysis was performed in a final volume of 20 µl containing 2 µl of cDNA, 0.2 µM of each primer, 0.03 µM of reference dye and 10 µl of Brilliant III Ultra Fast SYBR Green qPCR Master Mix (Agilent). Reactions were performed using an Mx3000P real-time PCR machine (Agilent) with the following program: 95°C for 3 min, then 40 cycles of 10 sec at 95°C and 20 sec at 60°C. Relative mRNA abundance of *Bc Tubulin* was normalized to the housekeeping gene *PUX1* (Windram *et al.*, 2012). The following primers were used: Bc Tub (Broad MIT ID: BC1G_00122) Fw: 5'-

TTCCATGAAGGAGGTTGAGG-3', Rv: 5'-TACCAACGAAGGTGGAGGAC-3'; PUX1 (At3g27310) Fw: 5'-AATGTTGCCTCCAATGTGTGA-3', Rv: 5'-TTTTTACCGCCTTTTGGCTAC-3'.

Methods S3 Metabolite analyses.

For metabolite analyses, an Acquity UPLC system coupled to a Synapt G2 QTOF mass spectrometer (Waters, Milford, MA) was employed. The entire system was controlled by Masslynx 4.1. The separation was performed in gradient mode on an Acquity BEH C18 column, 50 x 2.1 mm, 1.7 µm particle size (Waters) using a flow rate of 0.4 mL/min and mobile phases consisting of H_2O + formic acid 0.05% (phase A) and acetonitrile + formic acid 0.05% (phase B). The gradient program started at 2% B, increased linearly to 60% B in 4.0 min, then to 100% B in 2.0 min, the column was then washed with 100% B for 2.0 min before re-equilibration at initial conditions (2% B) for 2.0 min. The column temperature was maintained at 25°C throughout the run. The injection volume was 2 µL (partial loop with needle overfill mode). Mass spectrometric detection was performed in electrospray negative mode using a mass range of 50-600 Da. The MS capillary voltage was -2.0 kV, the cone voltage was -25V, the desolvation temperature and gas flow were 500°C and 800 L/h, respectively, the cone gas flow was 20 L/h, and the detector voltage was 2250 V. Accurate mass measurements were provided by infusing a 500 ng/mL solution of leucine-enkephalin through the LockSpray probe at a flow rate of 15 µL/min. The quantification of ICA was achieved by external calibration using calibration points at 5, 20, 100, 500 and 2000 ng/mL.

For analysis of leaf surface camalexin, *Botrytis cinerea*-infected or mock-treated leaves were immerged in 80% MeOH (2 mL/ 2 leaves) in 6-well plates and gently rotated for 30 sec. The solvent was collected in Eppendorf tubes and evaporated using a speed vac. The pellet was resuspended in 200 µl of 80% MeOH and transferred to vials for further LC-MS analysis. Quantification of camalexin was done according to (Balmer *et al.*, 2018). Values were normalized to the leaf surface and expressed as $\mu g/cm^2$. A total of 8 leaves (2 leaves from 4 plants) was used for each biological replicate.

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