Supplemental Figure 1. NaHER1 is systemically regulated after WOS treatment.
Patterns of local and systemic normalized signal intensity of NaHER1 transcript abundances (± SE, n=3) after WW and WOS treatments, and in control untreated plants extracted from a previously published microarray data set (Kim et al., 2011). WT leaves were wounded and immediately treated with 20 µL of water (WW), or 1:5 water-diluted oral secretions (WOS) from M. sexta larvae, and collected at the designated time points.
Supplemental Figure 2. Sequences, silencing construct and transgenic plant information. (A) Full length mRNA sequence of *NaHER1*; bold underlined sequence was used to create inverted repeat structure (hairpin) and independently silence *NaHER1* by VIGS and RNAi. (B) Physical map of pSOL8CBP plant binary transformation vectors used to generate irHER1 plants. The pSOL8CBP vector contains a strong constitutive cauliflower mosaic virus 35S (CaMV) promoter. cDNA fragment shown in (A) was cloned into the two multiple cloning sites in reverse orientation, which was separated by a pyruvate orthophosphate dikinase (pdk i3) intron to form an inverted repeat construct. (C) Southern blot analysis confirming the presence of a single T-DNA insertion in the *N. attenuata* genome of each transgenic line used in the study. A 10 μg aliquot of genomic DNA prepared from each of three independently transformed lines (irHER1-6/4, -8/6 and -9/6) was digested with XbaI restriction enzyme and separated by gel electrophoresis. Radioactively-labeled DNA fragment of the *hygromycin phosphotransferase* gene (hptII) was used as a hybridization probe. (D) Silencing efficiency of *NaHER1* in three independent *NaHER1*-silenced lines determined by qPCR.
**Supplemental Figure 3.** *NaHER1* silencing reduces emissions of several isoprenoid and phenylpropanoid/benzenoid volatiles from local WOS-treated *N. attenuata* leaves. Mean (± SE; n≥6) releases of volatile compounds from locally treated leaves of WT and irHER1 plants. A single local leaf in 35 d-old WT or irHER1 plants was mechanically wounded and treated with 20 µL of diluted OS from *M. sexta* (WOS). 18 h after treatment, volatiles were collected from the headspace of the same leaf for 7 h and analyzed by GC-MS. Different letters show significant differences between samples determined by one-way ANOVA, followed by a Fisher PLSD post hoc test (P ≤0.05). The volatile blends emitted from irHER1 leaves were strongly suppressed in the production of terpenoids (α-pinene, α-terpineol, β-pinene, D-limonene, (E)-α-bergamotene, α-duprezianene), and phenylpropanoid/benzenoids (benzaldehyd, benzyl alcohol). SE, standard error.
The sequence of the \textit{NaMYC2}. The cDNA sequence of bHLH-domain \textit{NaMYC2} was obtained after searching cDNA libraries of \textit{N. attenuata} using authentic \textit{A. thaliana} (AB000875) and \textit{N. benthamiana} \textit{MYC2} (GQ859153) sequences.
Supplemental Figure 5. Silencing of NaHER1 does not affect constitutive levels of phytohormones (ABA, JA, JA-Ile and SA) and defense metabolites (chlorogenic acid, caffeoylputrescine, dicafeoylspermidine, and total HGL-DTGs). Untreated rosette stage N. attenuata leaf samples were collected and analyzed by LC-MS/MS: ABA, JA, JA-Ile, SA (± SE, n=4) and HPLC: chlorogenic acid (CHA), caffeoylputrescine (CP), dicafeoylspermidine (DCS), HGL-DTGs (± SE, n=4), respectively. Leaves were divided into four equal parts during sampling and each part was analyzed separately. SE, standard error; FM, fresh mass; CHA eq., chlorogenic acid equivalents.
Supplemental Figure 6. cDNA fragment of *NaPYL4* gene. The cDNA sequence of a putative *N. attenuata* ABA receptor *NaPYL4* was obtained after searching cDNA libraries of *N. attenuata* using authentic *N. tabacum* PYL4 (AJ966358) sequence. The bold and underlined sequence was used to design VIGS construct and silence *NaPYL4*.
Supplemental Figure 7. *NaHER1* and *NaPYL4* silencing efficiency by VIGS.
Relative transcript abundances (± SE, n=4) of *NaHER1* and *NaPYL4* determined by qPCR. *NaHER1* and *NaPYL4* were silenced by VIGS method (see Material and Method for more details). Leaves were wounded and immediately treated with 20 µL of 1:5 water-diluted oral secretions (WOS) from *M. sexta* larvae. Samples were collected after 1 h and analyzed by qPCR. Asterisks represent significant differences determined by ANOVAs, followed by Fisher’s PLSDs post hoc test (* P ≤ 0.05), ** * P ≤ 0.001; SE, standard errors.
Supplemental Figure 8. ABA does not induce NaHER1 expression. Relative transcript abundances (± SE, n=5) of NaHER1 determined by qPCR. The leaves of *N. attenuata* (one per plant) were treated with 20 μL of 100 μM ABA in lanolin paste. Samples from treated leaves were collected at designated time points and analyzed by qPCR.
Supplemental Figure 9. The sequence of NaABA1 gene. The cDNA sequence of a putative N. attenuata NaABA1 was obtained after searching cDNA libraries of N. attenuata using authentic A. thaliana (AT5G67030) and Nicotiana plumbaginifolia (X95732) ABA1 sequence.
Supplemental Figure 10. The sequence of NaPDR12. The cDNA sequence of a putative *N. attenuata* ABA transporter NaPDR12 was obtained after searching cDNA libraries of *N. attenuata* using authentic *A. thaliana* PDR12 (NM_101421) sequence.
Supplemental Figure 11. Silencing of NaHER1 gene does not affect expression of the ABA biosynthetic gene NaABA1 or expression of the ABA transporter NaPDR12. Rosette stage *N. attenuata* plants were treated with WOS in zone 2. Samples were collected at designated time points, extracted and analyzed by real-time PCR for *NaABA1* and *NaPDR12* transcripts.
Supplemental Figure 12. *NaHER1* silencing alters ABA metabolism, JA and JA-Ile accumulation in detached leaf. Mean (± SE, n=4) levels of ABA metabolites, JA, JA-Ile, and SA in detached *N. attenuata* leaves collected at designated time points and analyzed by LC-MS/MS. Metabolite levels were compared by ANOVAs, followed by Fisher's PLSDs post hoc test (* P ≤ 0.05). SE, standard error.
Supplemental Figure 13. *NaHER1* silencing plants does not affect rosette diameter but delays flowering time and reduces seed capsule numbers in *N. attenuata*. Plants were maintained under continuous attack from *M. sexta* neonates (MS) or left untreated (Cont), and flower and seed capsule numbers (± SE, n=10) were counted at designated time points. Different letters show significant differences between samples determined by Mann-Whitney U test (*P* ≤0.05). Note that the flower numbers at the day 41 and 42 were present with their own y-axis.
Supplemental Figure 14. ABA affects JA accumulation of OS-elicited plants. Leaves of *N. attenuata* (one per plant) were mechanically wounded and immediately treated with 20 µL of diluted OS from *M. sexta* (WOS) or WOS supplemented with 100 mM ABA. Samples were collected after 4 d and analyzed by LC-MS/MS for phytohormone contents (± SE, n=4). Samples were compared by one-way ANOVA, followed by a Fisher PLSD post hoc test; different letters show significantly differences between samples (*P* ≤0.05). FM, fresh mass.
Supplemental Figure 15. Accumulation of individual HGL-DTGs in WT and irHER1 plants. Mean (± SE, n=4) accumulation of total and 10 individual HGL-DTGs determined by HPLC and LC-MS/MS, respectively. One leaf in each of the 47-d-old WT and irHER1 plants was treated with 20 µL diluted OS from *M. sexta* (WOS) and local and systemic samples [8th leaf above the local WOS leaf (sys+8 in figure)] were collected after 48 h and analyzed by HPLC and LC-MS/MS. Asterisks represent significant difference between WT and irHER1 plants determined by one-way ANOVA, followed by a Fisher PLSD post hoc test; * P ≤ 0.05, ** P < 0.01, *** P < 0.001. SE, standard error.
Supplemental Figure 16. Specific primer sequences used in qPCR (SYBR) analyses.

- NaEF1a forward primer: 5'-CCACACCTCCCACATTGCTGTC-3'
- NaEF1a reverse primer: 5'-CGCATGTCCCCTCACAGCAAAAC-3'
- NaMYC2 forward primer: 5'-CCTCCACCAGTCAAATCAAGA-3'
- NaMYC2 reverse primer: 5'-GACTCCCATTTTCACAGTTGCTTG-3'
- NaMYB8 forward primer: 5'-AACCTCAAGAAAATCCAGGACATAAC-3'
- NaMYB8 reverse primer: 5'-GATGAATGTGTGACCAAATTCC-3'
- NaHER1 forward primer: 5'-CCACGTCGCCACCTGTT-3'
- NaHER1 reverse primer: 5'-GAAACGCAGGTGTTGTTT-3'
- NaABA1 forward primer: 5'-GAAGGCAGCATCCTCGGT-3'
- NaABA1 reverse primer: 5'-CTTCAGAATTCTCAAGCTG-3'
- NaPDR12 forward primer: 5'-ACTGAAGAAGCTTTGAGGAAG-3'
- NaPDR12 reverse primer: 5'-GAATATTACAACACATCACC-3'
- NaPYL4 forward primer: 5'-CCATTAGCAGCACCACCTTAC-3'
- NaPYL4 reverse primer: 5'-GGAGCTTTATCTTTGGAG-3'