Table 1 Suppl. The primers used in the present study (F - forward primer, R - reverse primer).

Function	Primers name	Sequence (5'-3')
VpPDS degenerate primers	VpDPDSF	GCTKGARGCAAGRGATGT
	VpDPDSR	GCCTTTGACATRGCAATRAACACC
VpPDS RACE primers	VpPDS5'raceR	AGGGGGCACCTGATCGAGTTACTAC
	VpPDS3'raceF	CAGCCACCTTTCCACCTAGAACATC
VpPDS full - length primers	VpFPDSF	GATGAGCGTGCACGGGAGTGT
	VpFPDSR	GGCACAGAAGCCTTGAACAATA
NbGLO RACE primers	NbGLO15'raceR	GGCATCTGAGTTTGGTATTCATTCTC
	NbGLO25'raceR	GGCATCTGGTACTCGTAATCTCTGTC
NbGLO1 ORF primers	NbGLO1F	ATGGGGAACAACAAGAGAAAAG
	NbGLO1R	TTAGAACCTCTCCTGCAAATTAGGC
NbGLO2 ORF primers	NbGLO2F	ATGGGGAGAGGAAAGATAGAG
	NbGLO2R	CTACATTCTTTCATGTAGGTTTGGC
NbAP1 full - length primers	NbAP1F	ATGGGRAGAGGRAAAGT
	NbAP1R	GAMTCAAGATTTAGGTCAAG
TRV2::*VpPDS*	VpTPDSF	CTACCATGGGGCATTAAACTTCATTAACC
	VpTPDSR	CTAGGATCCCTTCAGTTTCCTGTCAAACC
TRV2::*VpPI*-ORF	VpTPIF	CTACCATGGTGTGGGATGCTAAACATGAGAACC
	VpTPIR	CTAGGATCCAAAGGCATCTGGGAGTTAT
TRV2::VpPI-3'UTR	VpTPI3'UTRF	AGCCATGGTAATACAAATCAGAGTAACC
	VpTPI3'UTRR	AAGGATCCGGTCCAATAGAGCCTCATCG
TRV2::*NbGLO1*-3'UTR	NbTGLO13'UTRF	AGCCATGGTAATTTGCAGGAGAGGTTC
	NbTGLO13'UTRR	AAGGATCCGCAGAATAGATAATGGTTATAG
TRV2::*NbGLO2*-3'UTR	NbTGLO23'UTRF	AGCCATGGTAGCATTATTAATTCCACT
	NbTGLO23'UTRR	AAGGATCCACTTAAAACCATAGGAAGAGCCC
pYL156	156F	TTACTCAAGGAAGCACGATGAGC
	156R	GAACCGTAGTTTAATGTCTTCGGG
RT-qPCR	Vp18sRTF	AAGACGAACAACTGCGAAAGC
	Vp18sRTR	AGCAACATCCGCCAATCC
	VpPDSRTF	CCAGTTGATATCCTGAAGC
	VpPDSRTR	GACATGTCAGCATATACAC
	NbactinF	GGCTTACATTGCTCTTGACTATGAAC
	NbactinR	ATCAGGCAGCTCGTAGCTCTTCT
	NbPDSRTF	CCAGTGGATATCTTGAAGC
	NbPDSRTR	GACATGTCAGCGTACACAC
	NbGLO1RTF	GGGAGAAGATTGTGGGATG
	NbGLO1RTR	GTGAGTCCATTATCAAGGG
	NbGLO2RTF	GAGGAAGTTTACCATCAAAG
	NbGLO2RTR	GCTAGTTCACCATATAACT
	NbDEFRTF	TGAACTCACTGTTCTTTGTGATGCT
	NbDEFRTR	CGTTTAGGCTTTCTCCCATCC
	NbTM6RTF	TCTGTAGCTGAGATACGTG
	NbTM6RTR	GCATAGTGGTTGTGTACTC
	NbAP1RTF	TCCTACTTCAACATCTTTCCTC
	NbAP1RTR	AGCTCATTCCTCCTTTCTTC

Table 2 Suppl. Gene silencing frequency in our virus-induced gene silencing (VIGS) analyses. ([1] - Occurrence of phenotypic variation; [2] - gene silencing extent of the potential direct target gene by a trigger sequence as indicated. For details, see Table 3 Suppl.).

	TRV2::VpPDS ORF *Viola*	*TRV2::VpPDS* ORF *Nicotiana*	*TRV2::VpPI* ORF	*TRV2::VpPI* 3'UTR	*TRV2::NbGLO2* 3'UTR	*TRV2::NbGLO1* 3'UTR
Plants treated	52	48	28	28	28	28
Plants with phenotypic variation	52	48	15	6	9	0
Plants without phenotypic variation	0	0	13	22	19	28
Gene silencing frequency[1] [%]	100	100	54	21	32	0
Gene silencing extent[2] [%]	96.70	73.91	70.74	68.68	74.11	55.86

Table 3 Suppl. The gene silencing extent of B-class MADS-box genes in *Nicotiana* (*red*: the gene silencing extent of the potential direct target gene by a trigger sequence as indicated).

	TRV2::VpPI ORF	*TRV2::VpPI* 3'UTR	*TRV2::NbGLO2* 3'UTR	*TRV2::NbGLO1* 3'UTR
NbGLO1	64.00±13.86 %	46.70±18.14 %	44.61±14.17 %	55.86±22.52 %
NbGLO2	70.74±19.56 %	68.68±10.74 %	74.11±11.55 %	22.97±6.50 %
NbDEF	45.68±18.79 %	44.10±16.36 %	47.72±14.49 %	17.47±8.38 %
NbTM6	26.72±18.58 %	7.74±12.30 %	14.47±17.93 %	4.20±2.13 %

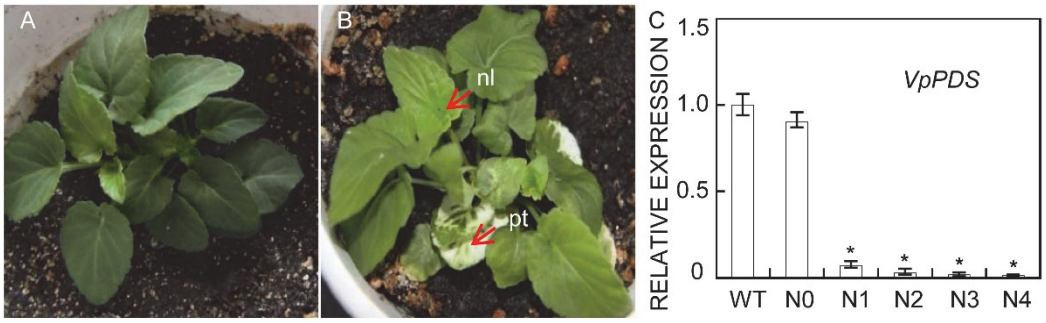
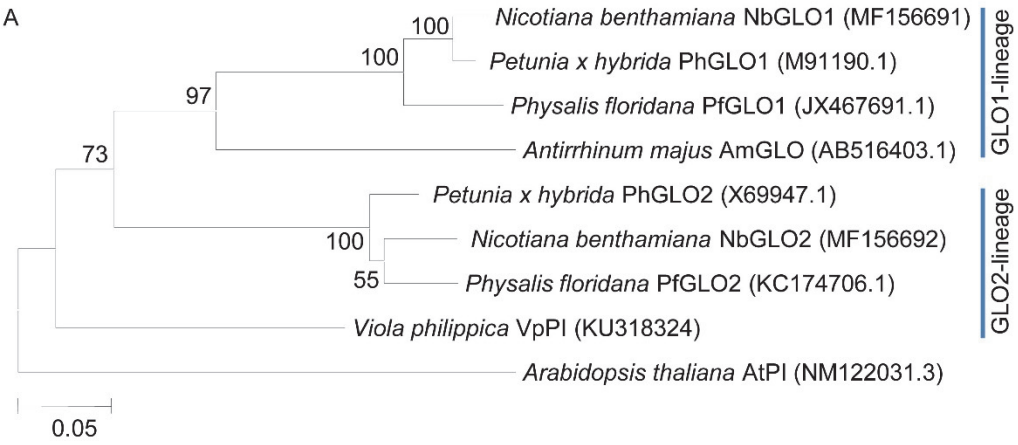


Fig. 1 Suppl. Gene silencing in *Viola philippica* using *VpPDS* as a trigger sequence. *A* - Wild type. B - Silencing endogenous *VpPDS* in the leaves of plants inoculated with *Agrobacterium tumefaciens* harboring *TRV2::VpPDS* (the *red arrows* indicate white patches on leaves; pt - photobleaching; nl - newly developed leaves). *C* - Relative expression of *VpPDS*; WT - wild type, NO - WT-like, no photobleaching, N1 to N4 - photobleaching leaves visually categorized according to the area of white patches. Means ± SDs, $n = 3$, * significant differences at $P \leq 0.01$.

A



B

	NbGLO2	NbGLO1	PhGLO2	PhGLO1	PfGLO2	PfGLO1
VpPI	68.1%	67.3%	69.5%	66.8%	68.6%	65.7%

Fig. 2 Suppl. Protein sequence identity between *VpPI* and other *PI-like* genes. *A* - Phylogenetic analysis of *PI-like* genes. The accession numbers are given in *brackets*. The *number* next to the nodes are bootstrap values. *B* - Protein sequence identity of VpPI with two GLOBOSA (GLO) paralogs in *Solanaceae*, which were duplicated to GLO1- and GLO2-lineages (Zhang *et al*. 2014b.) The VpPI shared a greater identity with the GLO2-lineage than with the GLO1-lineage. The results suggest that the VpPI is a putative ortolog of the GLO2-lineage, *i.e.*, NbGLO2 in *N. benthamiana*.

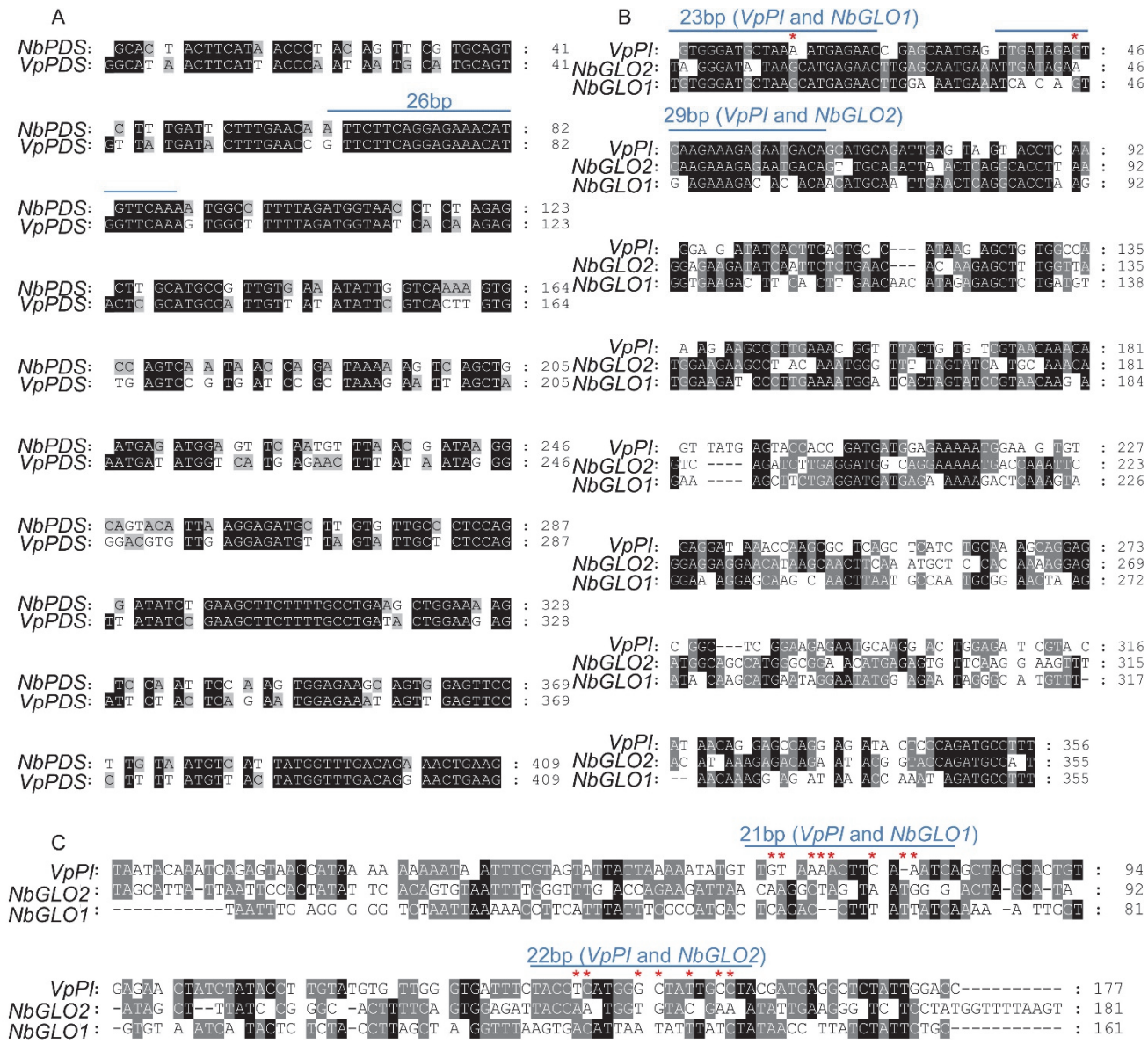


Fig. 3 Suppl. Sequence alignments between the trigger sequence and the target genes. *A* - The sequence alignment between *VpPDS* and *NbPDS*. *B* - The sequence alignment of *VpPI*-ORF (trigger sequence) and the corresponding fragments of *NbGLO1* and *NbGLO2*. *C* - The sequence alignment of the 3′UTR sequences of *VpPI*, *NbGLO1*, and *NbGLO2*. More than a 21-nucleotide stretch showing a 100 % identity between the trigger sequence and the target genes are indicated. The *red stars* indicate the mismatched bases. The trigger sequence length, sequence identity of the trigger sequence and target sequence, and other characteristics that potentially affect gene silencing are summarized in Table 1.