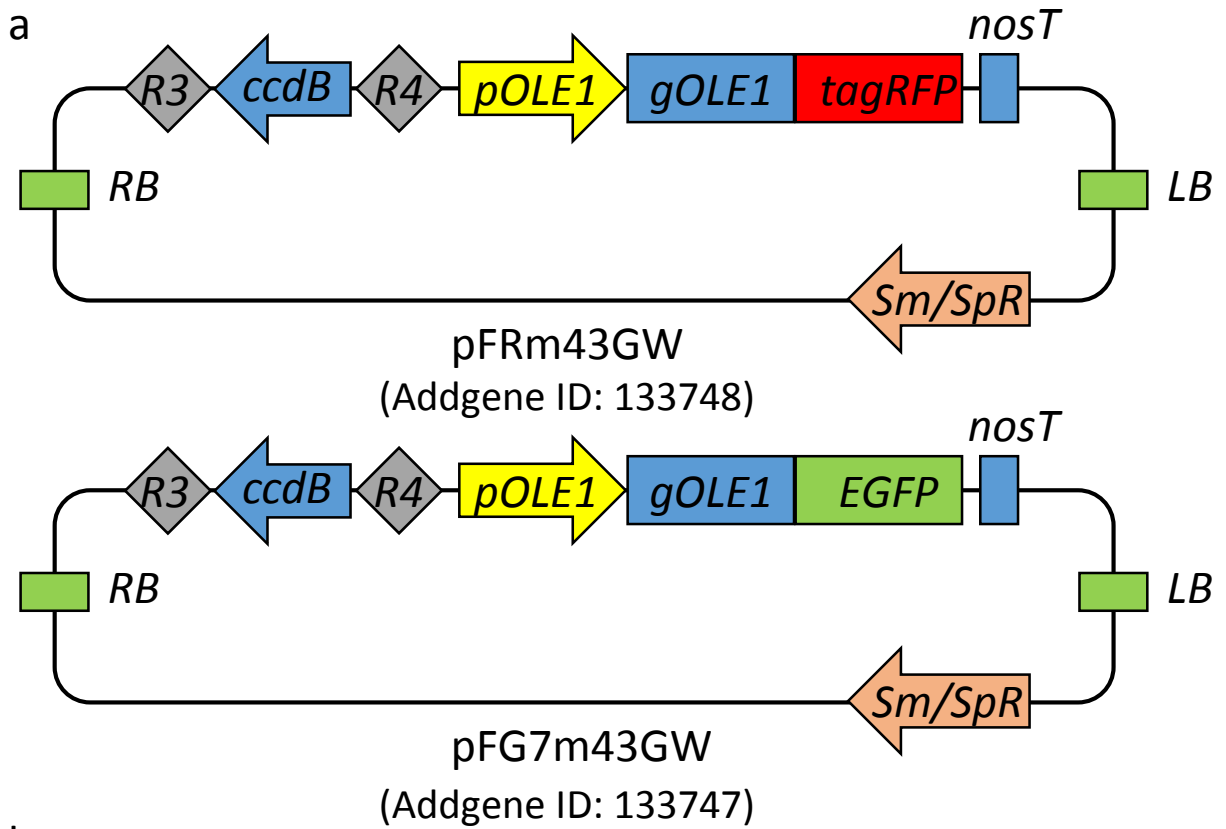
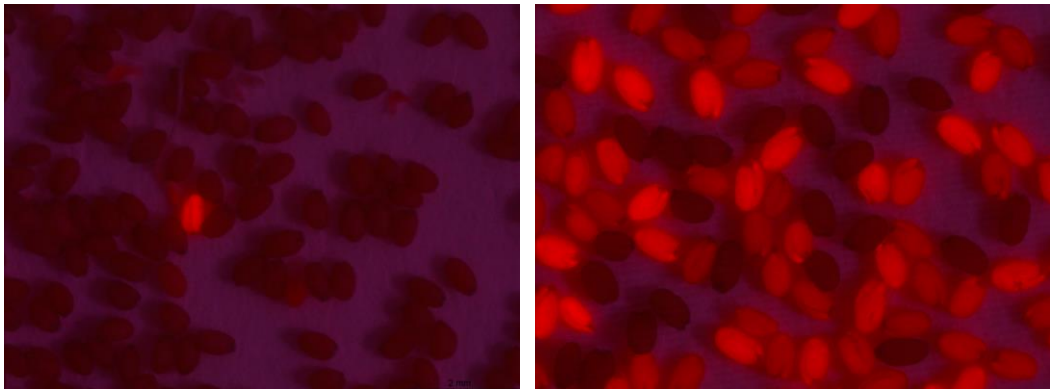


## Supplementary Figure 1



**b**



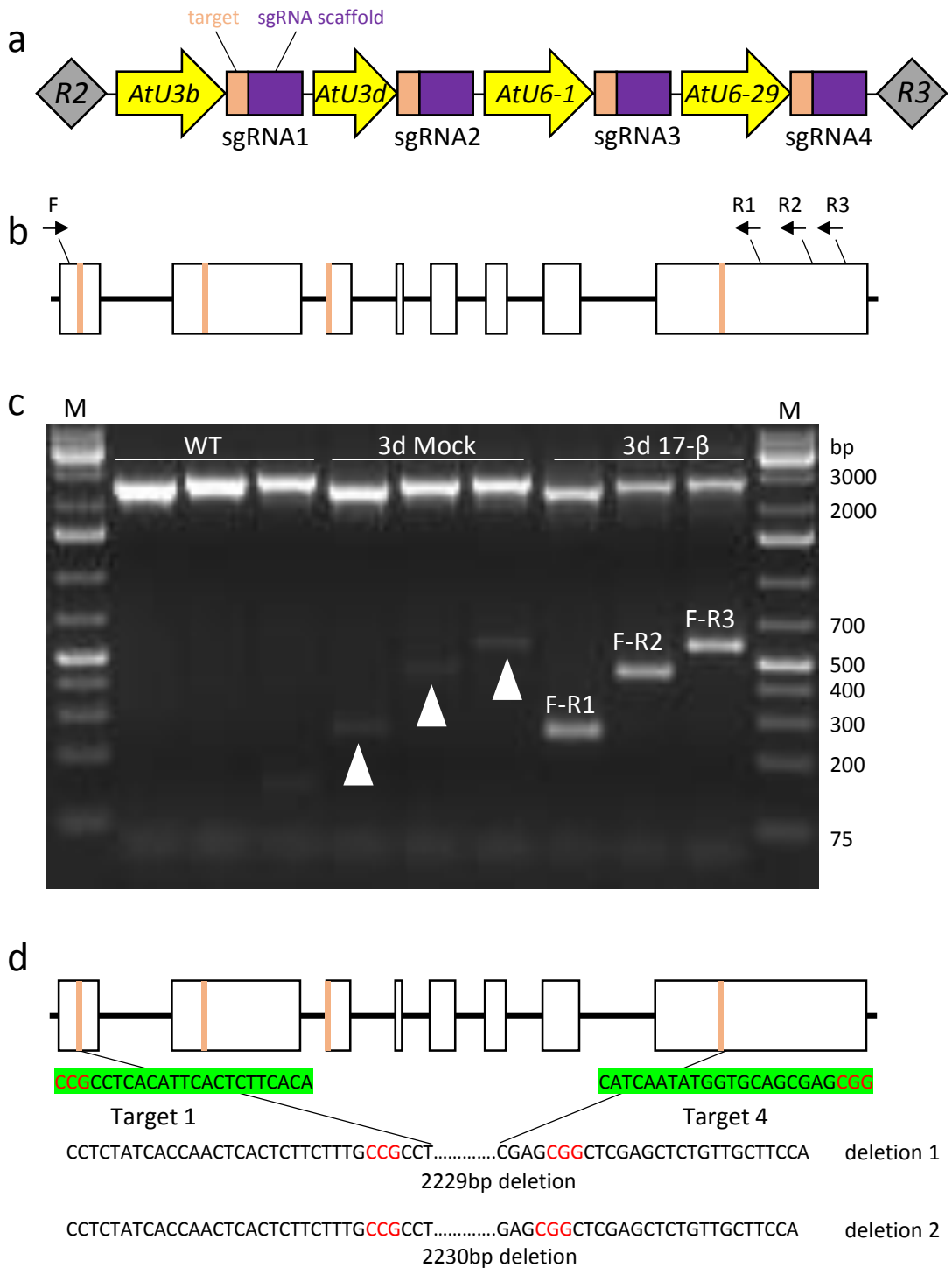
T1 screen

T2 screen

**Supplementary Figure 1 Non-destructive screening markers facilitate identification of transformed seeds.**

(a) Non-destructive fluorescent screening destination vectors generated in this study. (b) Examples of trans-pFRm43GW seeds screened under the fluorescence-binocular in the T1 (left) and T2 (right) generations.

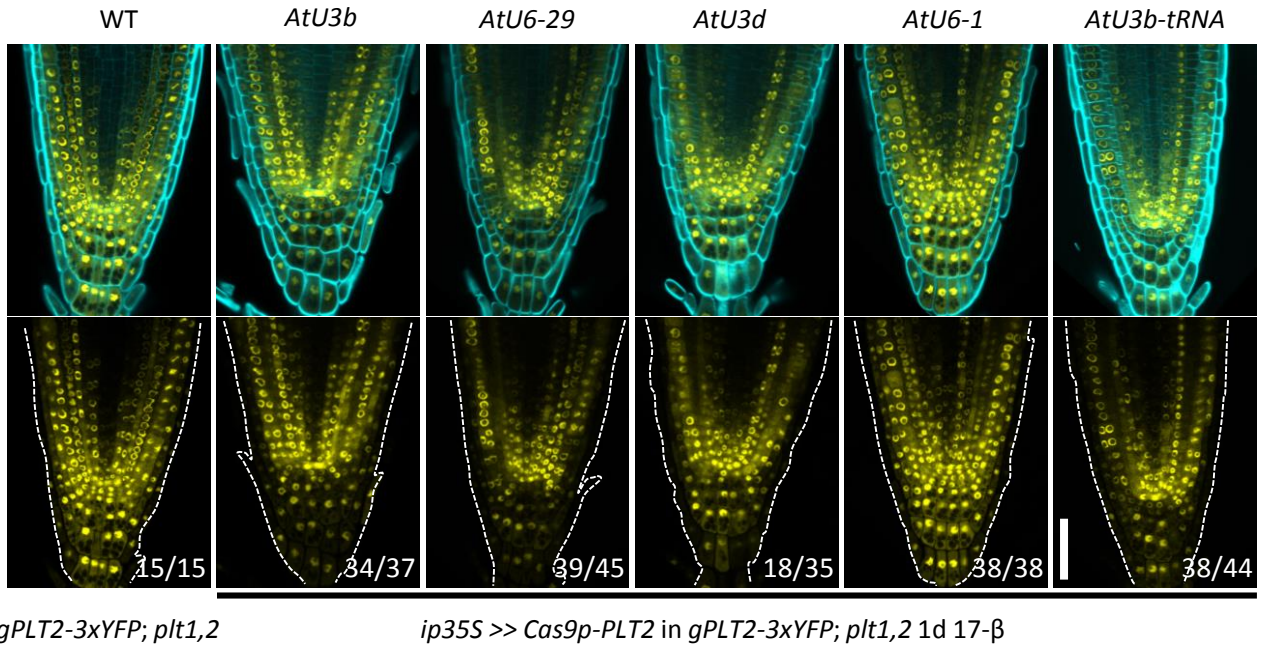
## Supplementary Figure 2



### Supplementary Figure 2 PCR genotyping of *PLT2* deletions.

(a) Tandem arrayed sgRNA expression cassettes. (b) The genomic structure of *PLT2*. Boxes indicate exons. Orange bars represent target sites in *PLT2*. Black arrows represent relative positions of the forward and reverse primers. (c) PCR detection of *PLT2* deletion in *ip35S>>Cas9p-PLT2; gPLT2-3xYFP; plt1,2* T1 seedlings after 3 days of treatment (in 6 day-old plants). Pooled DNA was isolated from 2cm root segments below the hypocotyl of 10 seedlings. Three primer pairs were used. There were no detectable truncated bands in 7-day old *gPLT2 3xYFP; plt1,2*, while weak truncated bands were detected in mock treated seedlings (white arrowhead), probably due to weak leakiness of *ip35S* in certain roots or cells. Note that although four sgRNAs were used to target *PLT2*, only one truncated band was detected with each primer pair. (d) Sequencing of truncated bands from primer pair F-R3 confirmed deletion between the 1<sup>st</sup> and 4<sup>th</sup> *PLT2* target sites (letters in red represent protospacer adjacent motif, PAM). To determine the deletion types, the truncated band was not directly used for sequencing but cloned into *pDONR 221*. Two deletion types were found in 4 sequenced recombinant vectors.

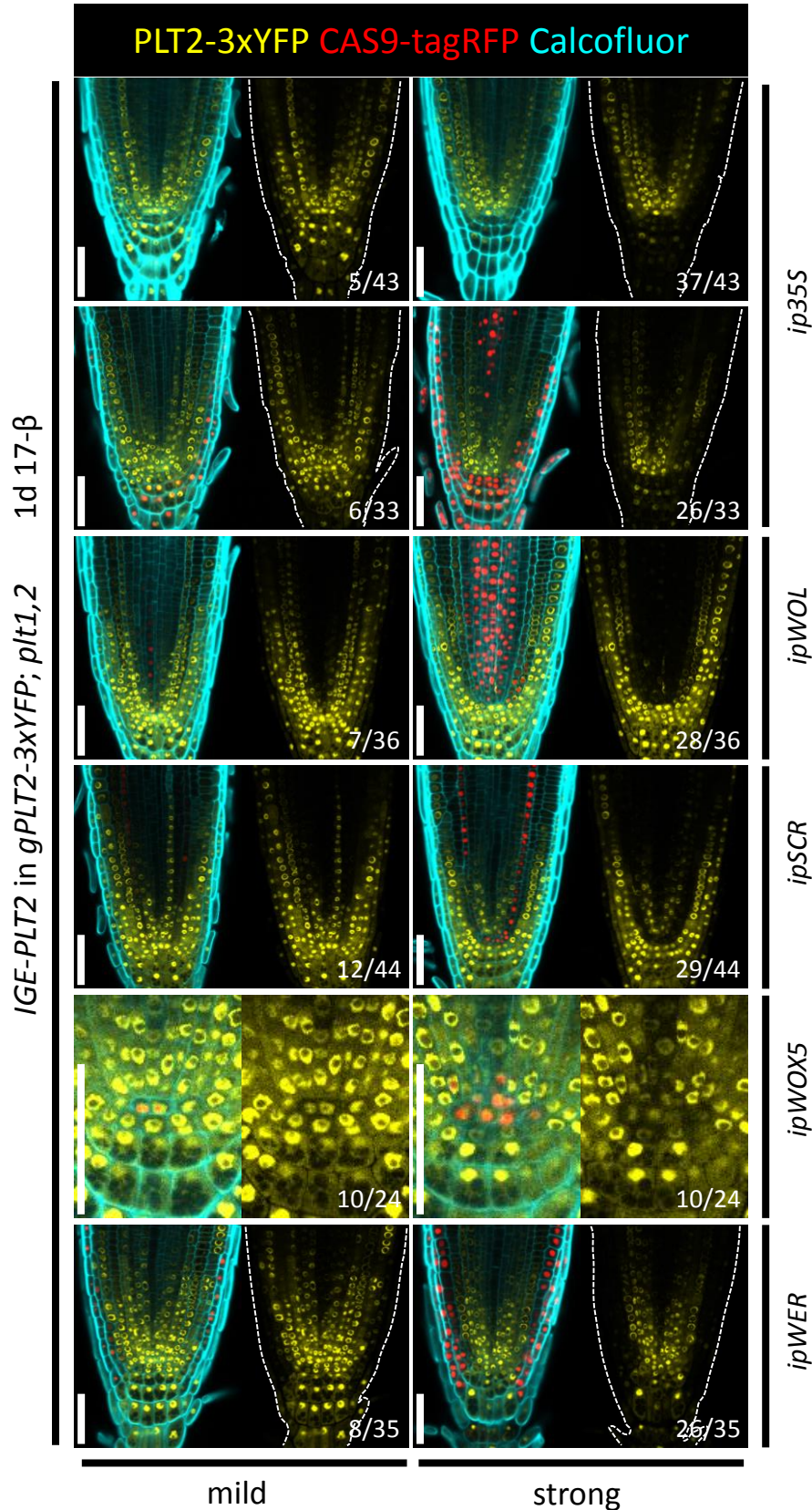
## Supplementary Figure 3



### Supplementary Figure 3 sgRNA promoter identity affects editing efficiency in *Arabidopsis* roots.

For each construct, the indicated sgRNA promoter was used to drive transcription of sgRNA1, while *ip35S* was used to guide *Cas9p* transcription. *AtU3b* and *AtU6-29* showed the best editing efficiency in T1 seedlings after one day of induction. Transcription of tRNA together with sgRNA1 under the *AtU3b* promoter also resulted in efficient *PLT2* editing. WT is the 7-day old *gPLT2-3xYFP; plt1,2*. White dotted lines mark the RM outlines. Cell walls are highlighted by calcofluor. Numbers indicate the frequency of similar results in the independent T1 samples analyzed. Scale bar, 50 μm.

## Supplementary Figure 4

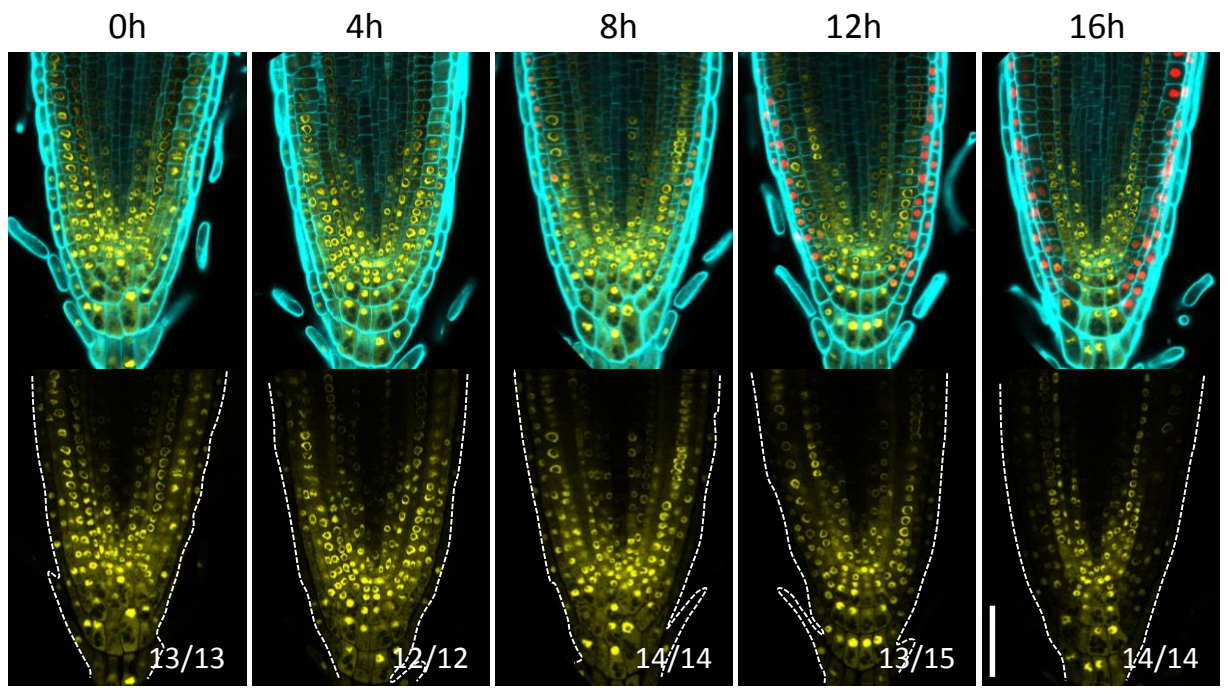


### Supplementary Figure 4 IGE-mediated genome editing correlates with Cas9 expression.

After one day of induction, IGE performance on *PLT2* editing under different inducible promoters was classified into two categories. In the mild category, Cas9p/Cas9p-tagRFP expression tends to be weak and narrow, resulting in narrow domains of moderately decreased YFP signal. In the strong category, Cas9p-tagRFP expression was strong and broad, with strongly and broadly reduced YFP fluorescence. In the uppermost panel, Cas9p was used without a tag. White dotted lines mark the RM outlines. Cell walls are visualized by calcofluor. Numbers indicate the frequency of similar results in the T1 samples analyzed. Scale bars, 50  $\mu\text{m}$ .



## Supplementary Figure 5

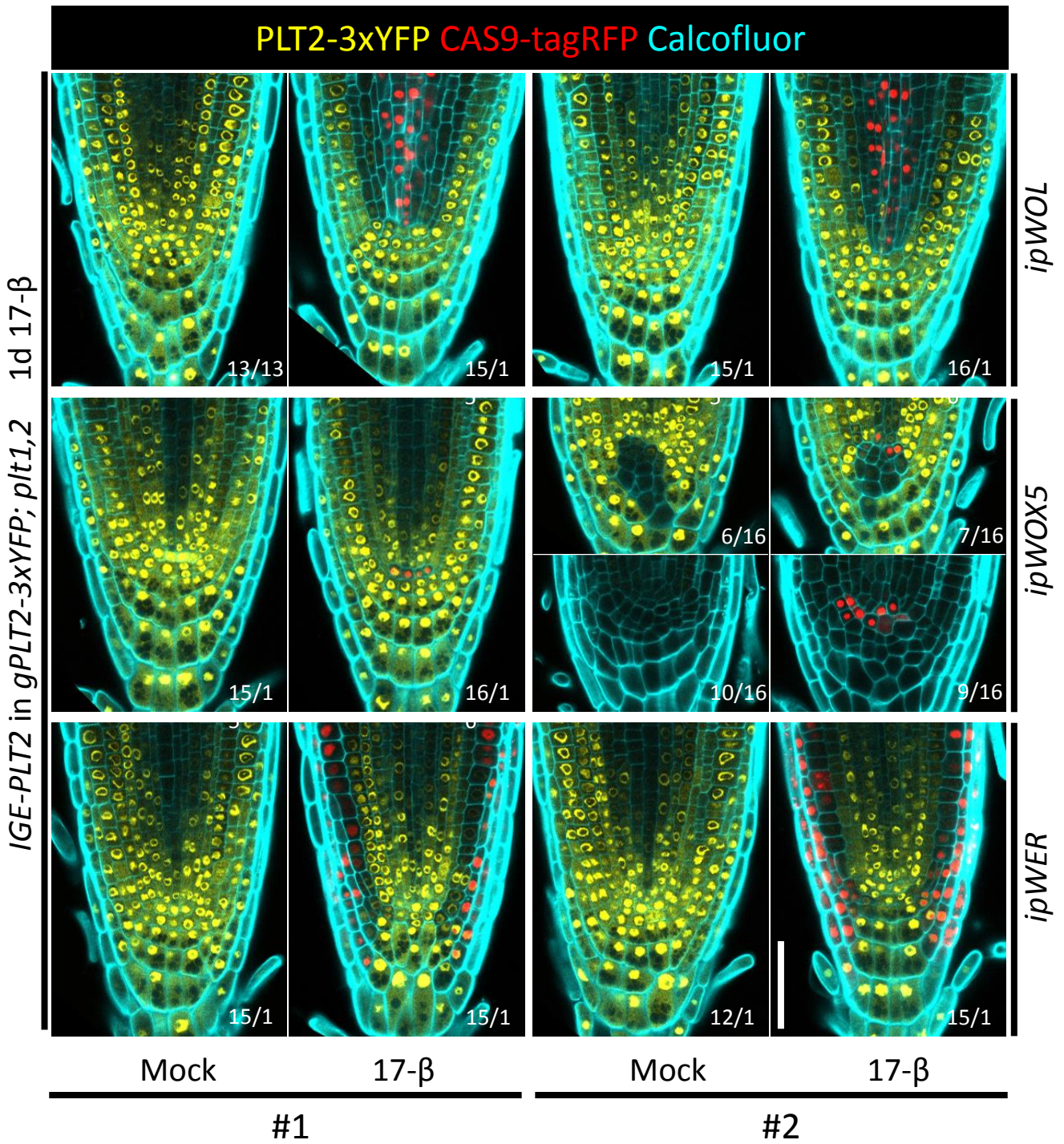


*ipWER* >> *Cas9p-tagRFP-PLT2* in *gPLT2-3xYFP; plt1,2*,

### Supplementary Figure 5 IGE system enables real time observation of genome editing.

To monitor *PLT2* editing dynamics, a time-course 17- $\beta$  induction was conducted to *ipWER* >> *Cas9p-tagRFP-PLT2* in *gPLT2-3xYFP; plt1,2* (T2 generation, #1). The reduction of *PLT2-3xYFP* expression was first detected after 12 hours of induction and became obvious with 16 hours of induction. The editing activity was gradually spread inwards, likely due to the radial diffusion of 17- $\beta$  within *ipWER* domain. White dotted lines mark the RM outlines. Cell walls are visualized by calcofluor. Numbers indicate the frequency of observed phenotype within given induction duration. Scale bar, 50  $\mu$ m.

## Supplementary Figure 6

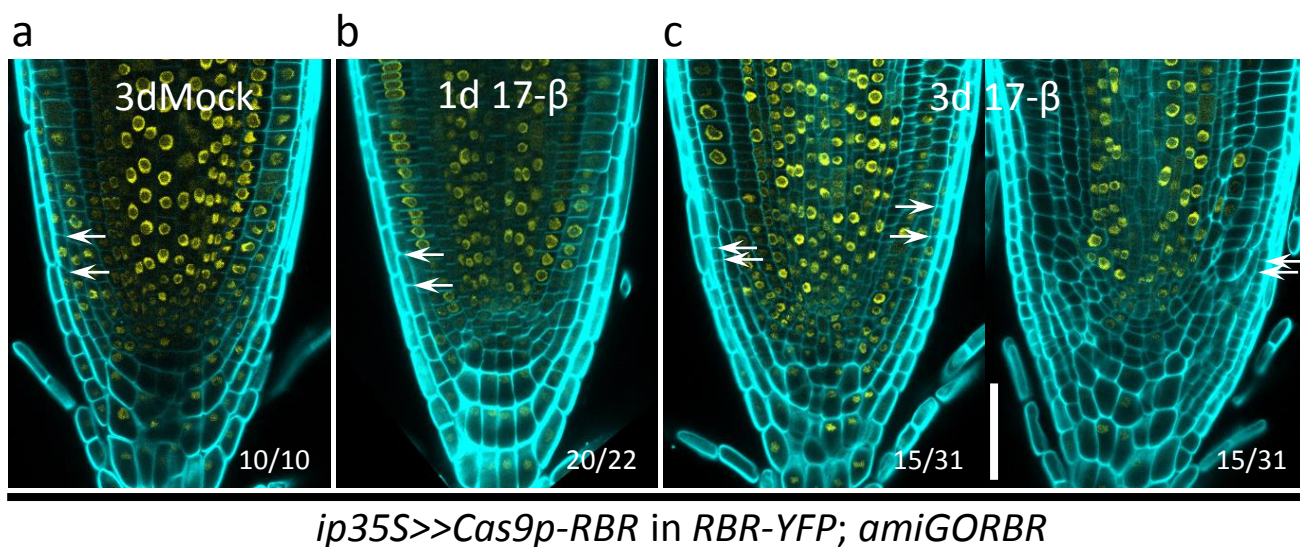


### Supplementary Figure 6 The capacity of conditional genome editing by IGE system is inherited.

For each construct, two independent transgenic T2 lines were randomly selected and checked. Representative images are shown. Note that the second *ipWOX5*>>*Cas9p-tagRFP-PLT2* line was leaky: roots displayed a similar phenotype with/without induction. Cell walls are marked by calcofluor. Numbers represent the frequency of the observed phenotype in analyzed T2 samples. Scale bar, 50 μm.



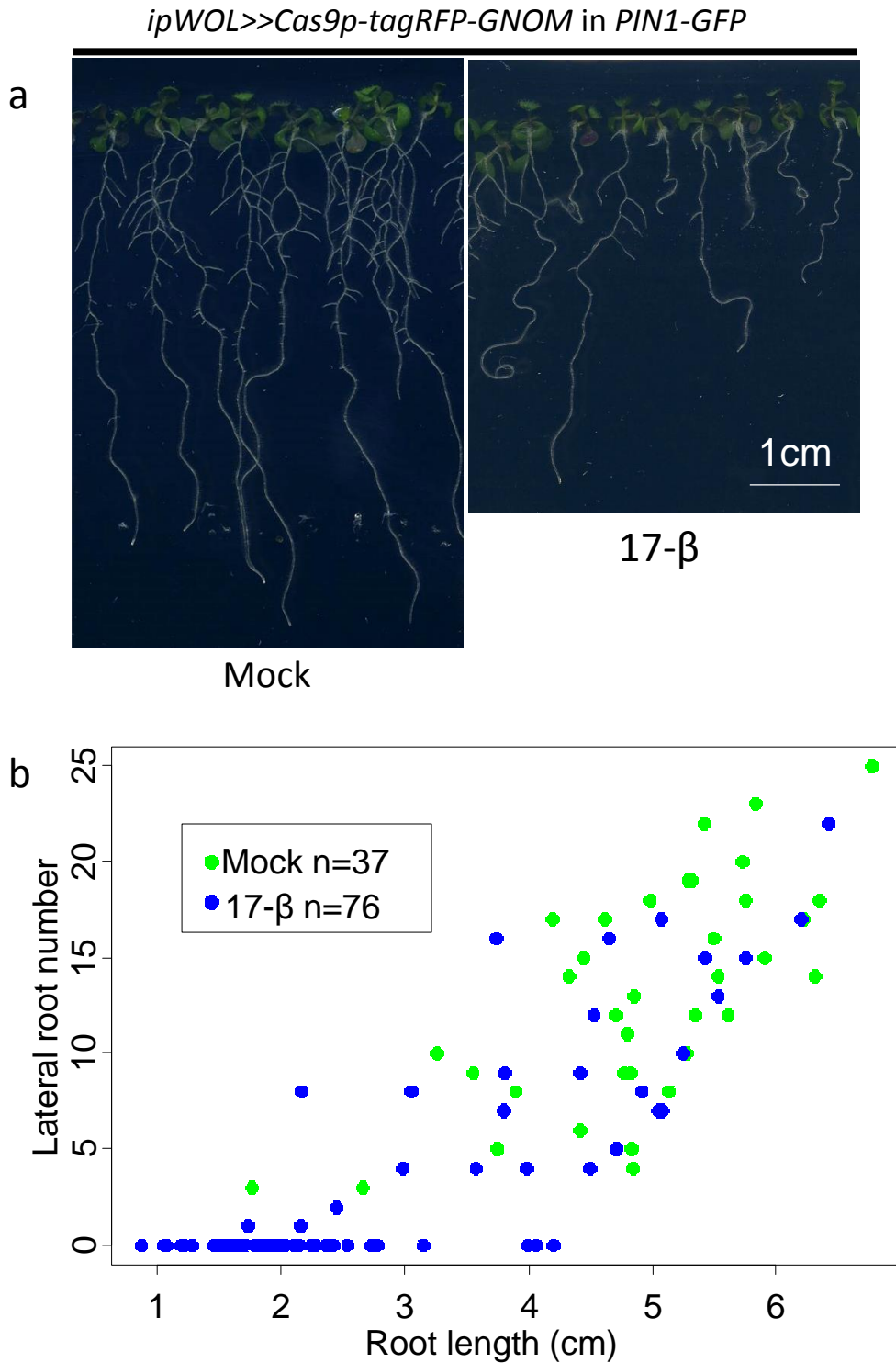
## Supplementary Figure 7



### Supplementary Figure 7 RBR functions cell-autonomously in the RM.

(a) A three-day mock treatment of *ip35S>>Cas9p-RBR* in *RBR-YFP; amiGORBR*. (b) A one-day induction caused a reduced RBR-YFP signal mainly in the root cap region without an obvious phenotype. (c) Inducing *RBR* editing with *ip35S* typically led to LRC overproliferation (white arrows) without affecting the YFP signal in other domains after a 3-day induction. In some cases, both wild type cells and RBR-knockout cells were seen on the same root (left in c). Cell walls are visualized by calcofluor. Numbers indicate the frequency of the observed phenotype in independent T1 samples. Scale bar, 50 μm.

## Supplementary Figure 8

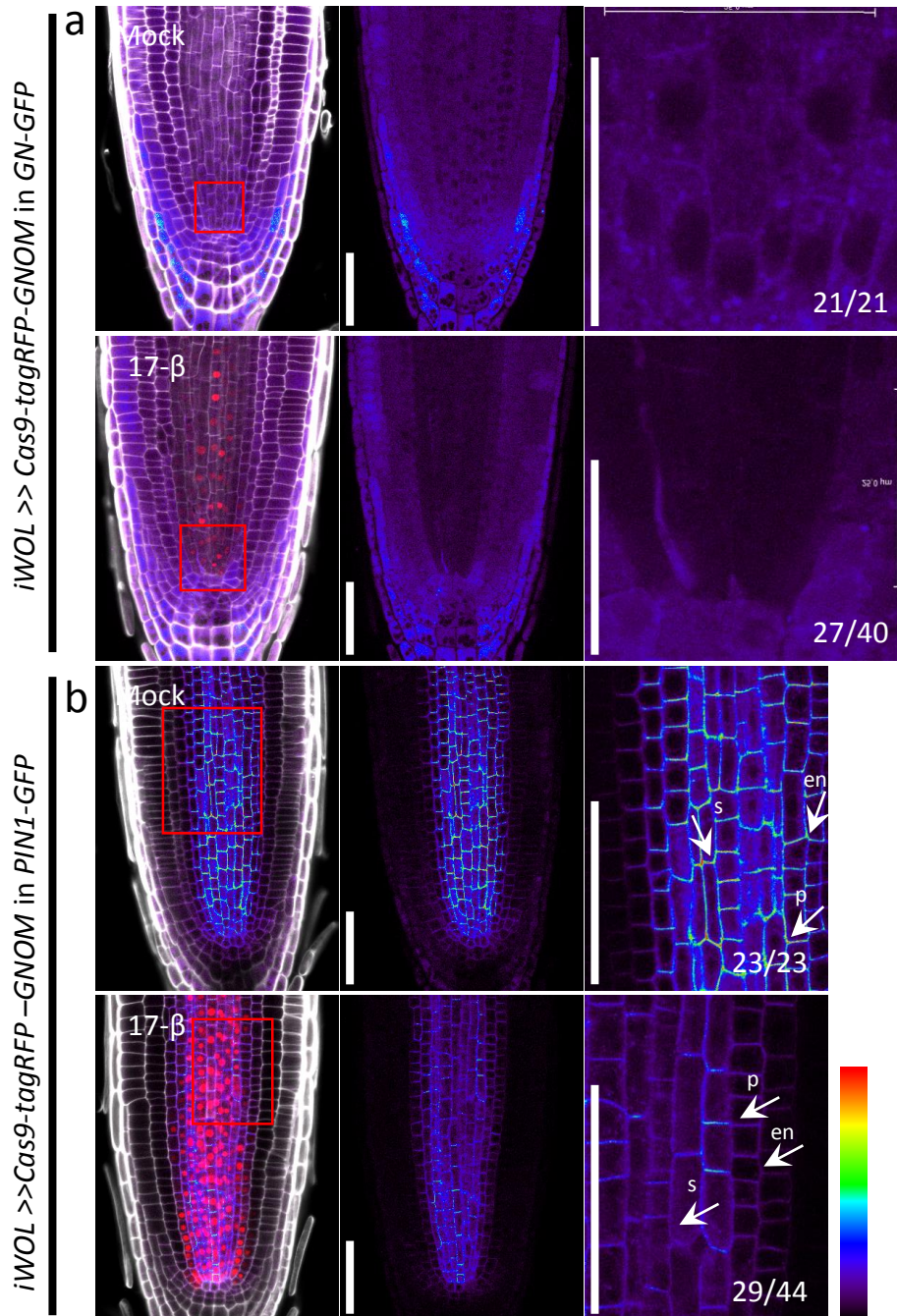


**Supplementary Figure 8 Post-embryonically inducing *GNOM* editing recapitulates the phenotypes of the *gnom* mutant.**

(a) Plants with *ipWOL>>Cas9p-tagRFP-GNOM*; *PIN1-GFP* ten days after germination on mock or 17-β plates. Inducing *GNOM* editing led to shorter roots, agravitropic growth and decreased lateral root (LR) numbers. Adventitious roots from the hypocotyl were frequently found, but these roots were not counted in LR quantification. For each independent root, LR number and root length is quantified in (b). Scale bar, 1 cm.



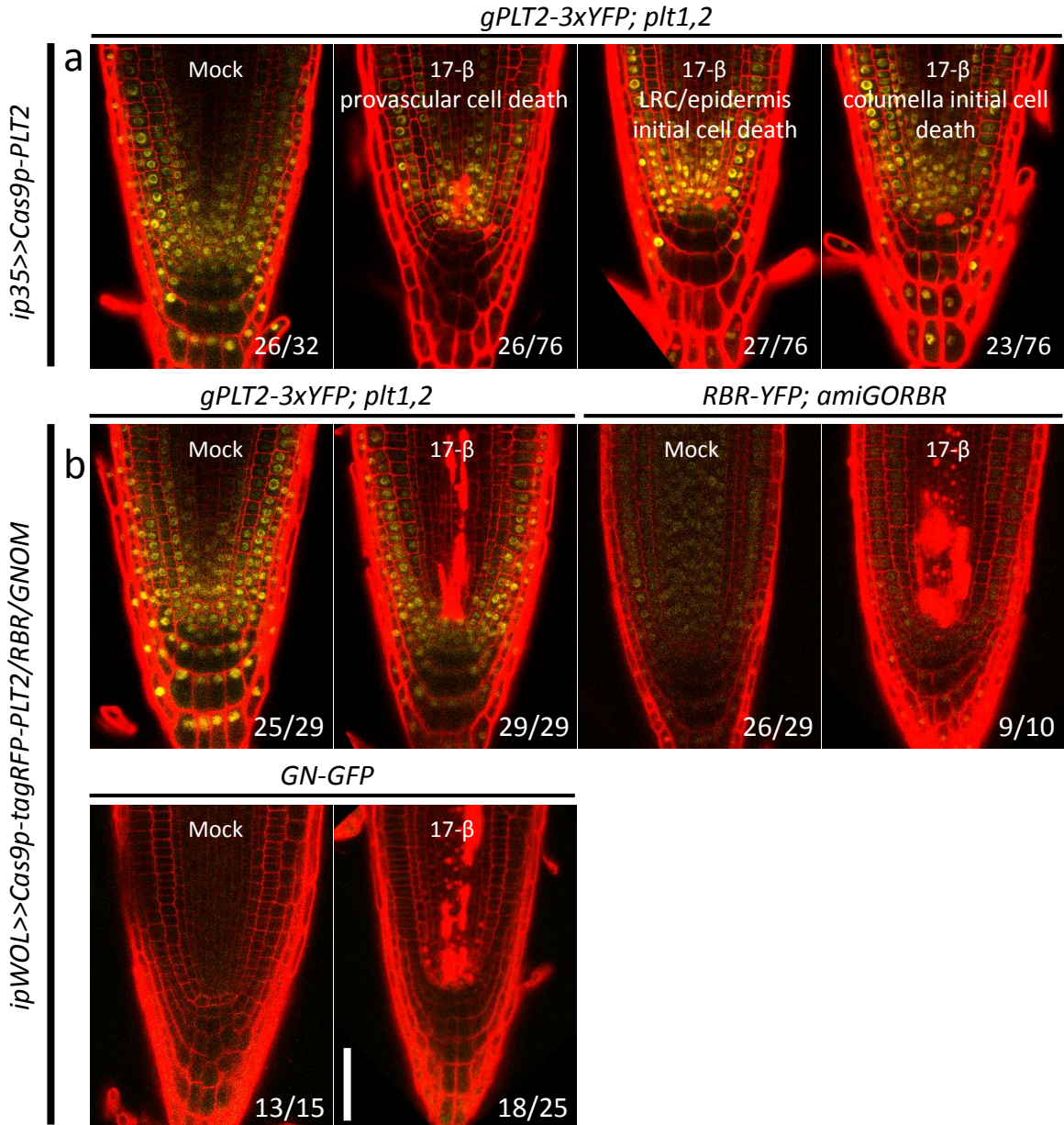
## Supplementary Figure 9



### Supplementary Figure 9 GNOM is required for PIN1 polarity and expression.

(a) *GNOM* expression disappeared from the vasculature after a 6-day induction of IGE targeting *GNOM*. Due to the weak GFP signal, only roots showing a clear loss of GFP signal were included in quantification. (b) A three-day induction of *ipWOL >> Cas9p-tagRFP-GNOM*; *PIN1-GFP* resulted in loss of polarity and decreased expression of *PIN1-GFP* in the endodermis (en), pericycle (p) and stele (s) (white arrows). Right panels are magnified images of the regions marked with a red box in the left panels. Cell walls are marked by calcofluor. Numbers indicate the frequency of the observed phenotype in independent T1 samples analyzed. Scale bar in right panels of a, 25  $\mu\text{m}$ ; others, 50  $\mu\text{m}$ .

## Supplementary Figure 10

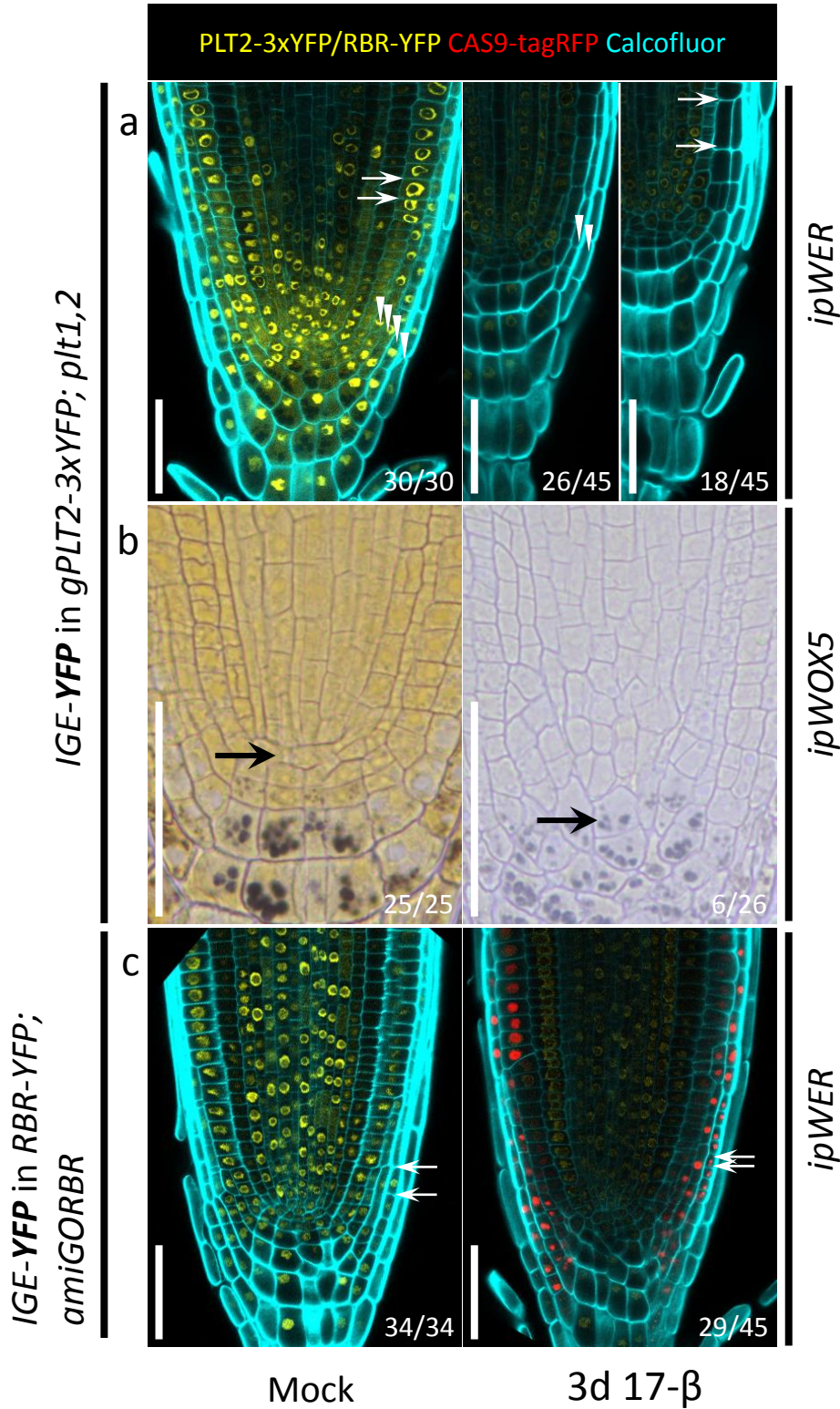


### Supplementary Figure 10 Cas9p-mediated genome editing in proximal stem cells induces cell death.

(a) Stem cell death surrounding the QC was observed after one day of *ip35S>>Cas9p-PLT2* induction. Based on cell types, the cell death response is classified into three categories: provascular cell death, LRC/epidermis initial cell death and columella initial cell death. Samples were counted twice if they had cell death in different domains. (b) Cell death of provascular cells and early descendants was induced after one day of induction of *ipWOL>>Cas9p-tagRFP-PLT2/RBR/GNOM*. Cell walls are highlighted by propidium iodide (PI). Under PI detection settings, Cas9p-tagRFP is also visible. Numbers indicate the frequency of the observed phenotype in independent T1 samples analyzed. Scale bars, 50  $\mu$ m.



# Supplementary Figure 11

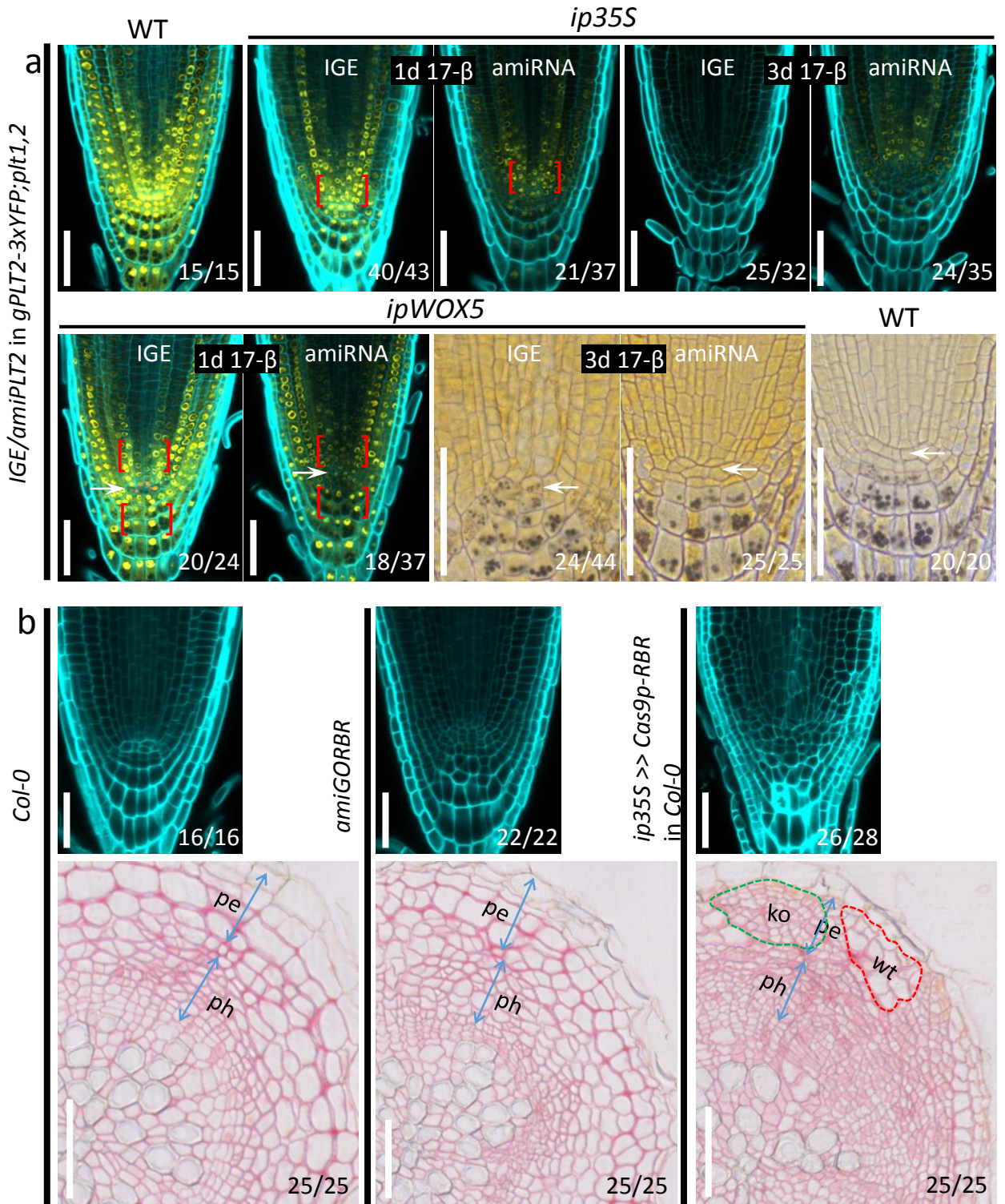


**Supplementary Figure 11 A single IGE construct targeting a gene encoding a fluorescent reporter has the potential to disrupt different transgene targets.**

(a) Editing *YFP* instead of *PLT2* in the *ipWER* expression region caused changes similar to direct *PLT2* editing. The RM had fewer LRC layers (white arrowheads), as well as premature expansion of epidermal cells and a broad, faint YFP signal. The Cas9p-tagRFP signal is frequently invisible. (b) Editing *YFP* led to QC (black arrow) differentiation at a lower frequency. (c) Targeting the *YFP* of *RBR-YFP* in the LRC led to LRC overproliferation, similar to editing *RBR*. However, the YFP signal outside *ipWER* expression region was also hampered by an unknown mechanism, unlike when editing *RBR*. White arrows mark the neighboring cell walls in a and c. The same construct was used in a and c. Cell walls are highlighted by calcofluor. Numbers indicate the frequency of the observed phenotype in independent T1 samples analyzed. Scale bars, 50  $\mu$ m.



# Supplementary Figure 12



**Supplementary Figure 12 Comparison of IGE system with inducible amiRNA.**

**(a)** IGE-PLT2 displays more specific and stronger *PLT2*-YFP downregulation than amiPLT2. After a one-day induction, *ip35S*>>*amiPLT2-1*; *gPLT2-3xYFP;plt1,2* and *ipWOX5*>>*amiPLT2-1*; *gPLT2-3xYFP;plt1,2* showed a broader reduction of the YFP signal, particularly in the bracketed regions where no inducible promoter activity was found. Conversely, induced *PLT2* editing caused very local loss of the YFP signal. After a three-day induction, the YFP signal is still visible in most of *ip35S*>>*amiPLT2-1*; *gPLT2-3xYFP; plt1,2* transformants but not in *ip35S*>>*Cas9p-PLT2*; *gPLT2-3xYFP;plt1,2* transformants. There was no QC differentiation in *ipWOX5*>>*amiPLT2-1*; *gPLT2-3xYFP; plt1,2* roots. WT here means 7-day old *gPLT2-3xYFP; plt1,2*. White arrows mark the QC. **(b)** Comparison of the RM and root secondary growth of *Col-0*, *35S:amiGORBR* and *ip35S*>>*Cas9p-RBR*. Inducing *RBR* editing (germination and six days of growth on 17- $\beta$  plates) resulted in more excessive cell divisions in the LRC than was seen in *amiGORBR* roots (germination and six days of growth on 17- $\beta$  free plates). Furthermore, *RBR* editing caused cell overproliferation in secondary tissues such as phloem (ph) cells and the periderm (pe), which was not observed in *amiGORBR* roots. The knockout (ko) sectors (green dotted line) were frequently accompanied by WT sectors (red dotted line), which can be regarded as an internal control. Cell walls are marked by calcofluor. Numbers indicate the frequency of observed phenotype in independent samples analyzed. Scale bars, 50  $\mu$ m.

**Supplementary Table 1 Quantification of fully differentiated RM after 10 days induction.**

1st BOX	2nd BOX	3rd BOX	Differentiated RM after 10d 17-β induction. Two repeats	
p1R4-35S-XVE	p221z-CAS9p	p2R3z-PLT2-AtU3b-sgRNA1	31/47 (66.0 %)	25/41 (61.0 %)
		p2R3z-PLT2-AtU3d-sgRNA1	17/32 (53,1 %)	20/48 (41,7 %)
		p2R3z-PLT2-AtU6-1-sgRNA1	0/29 (0.0 %)	0/43 (0.0 %)
		p2R3z-PLT2-AtU6-29-sgRNA1	15/23 (65.2 %)	22/34 (64.7 %)
		p2R3z-PLT2-AtU3b-tRNA-sgRNA1	20/34 (58.8 %)	25/31 (80.6 %)
		p2R3z-PLT2-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	17/32 (53.1 %)	25/35 (71.4 %)
	p221z-CAS9p-taqRFP	p2R3z-PLT2-AtU3b-sgRNA1	21/32 (65.6 %)	23/39 (59.0 %)
	p221z-dCAS9p	p2R3z-PLT2-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	0/32 (0.0 %)	0/41 (0.0 %)
	p221z-AtMIR390-PLT2-1	p2R3z-nosT2	0/29 (0.0 %)	0/32 (0.0 %)
	p221z-AtMIR390-PLT2-2	p2R3z-nosT2	0/24 (0.0 %)	0/37 (0.0 %)

**Supplementary Table 2 Primer list in this study.**

Primer name	sequence(5'-3')	purpose
attB1-CAS9p-T35s-F	GGGGACAAGTTTGTACAAAAAAGCAGGCTCGATGGCTCCT AAGAAGAAGCG	For cloning CAS9p with T35s terminator into 2nd BOX
attB2-CAS9p-T35s-R	GGGGACCACTTTGTACAAGAAAGCTGGGTGGTCACTGGA TTTTGGTTTTAGG	
attB2-ccdB-F	GGGGACAGCTTCTTGTACAAAGTGGAACTCGAGAGACCT CTGAAGTGG	clone Bsa I-ccdB-Bsa I into 3 box
attB3-ccdB-R	GGGGACAACCTTTGTATAATAAAGTTGAACCGCGAGACCCA CGCTCAC	
PLT2-TG1-gRT#+	TGTGAAGAGTGAATGTGAGG	clone 4 sgRNA expression cassettes targeting PLT2
PLT2-TG1-AtU3bT#-	CCTCACATTCCTTCCATGACCAATGTTGCTCC	
PLT2-TG2-gRT#+	ATAAGGTACGAGGTTGTGAT	
PLT2-TG2-AtU3dT#-	ATCACAACCTCGTACCTTATTGACCAATGGTGCTTTG	
PLT2-TG3-gRT#+	TTAGATAACTAAGTACGAGAG	
PLT2-TG3-AtU6-1T#-	TCTCGTAGTTAGTTATCTAACAATCACTACTTCGTCT	
PLT2-TG4-gRT#+	CATCAATATGGTGCAGCGAG	
PLT2-TG4-AtU6-29T#-	CTCGCTGCACCATATTGATGCAATCTCTTAGTCGACT	
dCas9p-D10A-F	TACTCCATCGGCCTCgcgATCGGCACCAACAGC	dCas9 cloning
dCas9p-H840A-R	GACTGAGGAACAATcgGTCGACGTCGTAGT	
attB1-gPLT2-F	GGGGACAAGTTTGTACAAAAAAGCAGGCTCGATGAATTCT AACAACTGGCTC	PCR detection of PLT2 deletion from genome, and subsequent cloning into pDONR221z for sequencing
attB2-gPLT2-R1	GGGGACCACTTTGTACAAGAAAGCTGGGTGGAATCATGA TACTGAGAGAT	
attB2-gPLT2-R2	GGGGACCACTTTGTACAAGAAAGCTGGGTGGAGCTTGAC CCAATACCAAT	
attB2-gPLT2-R3	GGGGACCACTTTGTACAAGAAAGCTGGGTGGATCCTTGA GCAGACTCTCC	
amiPLT2-1-F	TGTATGATGATCCCCGATTTGCTGATGATGATCACATTCC TTATCTATTTTTTCAGCAAATCGTGGGATCATCA	amiPLT2-1 cloning
amiPLT2-1-R	AATGTGATGATCCCACGATTTGCTGAAAAAATAGATAACG AATGTGATCATCATCAGCAAATCGGGGGATCATCA	
amiPLT2-2-F	TGTATGATCGGTGTGATGATCCCCGATGATGATCACATTC GTTATCTATTTTTTCGGGGATCATAACACCGATCA	amiPLT2-2 cloning
amiPLT2-2-R	AATGTGATCGGTGTTATGATCCCCGAAAAAATAGATAACG AATGTGATCATCATCGGGGATCATCACACCGATCA	
PLT2-TG1-AtU3dT#-	CCTCACATTCCTTCCATGACCAATGGTGCTTTG	sgRNA promoter comparison
PLT2-TG1-AtU6-1T#-	CCTCACATTCCTTCCACAATCACTACTTCGTCT	
PLT2-TG1-AtU6-29T#-	CCTCACATTCCTTCCACAATCTCTTAGTCGACT	
YFP-gRT	CCCATCTGGTTCGAGCTGGA	YFP targeting
AtU3b-YFP	TCCAGCTCGACCAGGATGGGTGACCAATGTTGCTCC	
RBR-TG1-gRT#+	TCAGCAAGCATGTCTAACAT	For cloning 4 sgRNA expression cassettes targeting RBR
RBR-TG1-AtU3bT#	ATGTTAGACATGCTTGTGATGACCAATGTTGCTCC	
RBR-TG2-gRT#+	GTC AAGGCTGGATCTG TACT	
RBR-TG2-AtU3dT#	AGTACAGATCCAGCCTTGACTGACCAATGGTGCTTTG	
RBR-TG3-gRT#+	TATCCTCAACTCATCTTCTG	
RBR-TG3-AtU6-1T#	CAGAAGATGAGTTGAGGATACAATCACTACTTCGTCT	
RBR-TG4-gRT#+	TATGACAGTCTGAGCCACT	
RBR-TG4-AtU6-29T#	AGTGGCTCAGGACTGTCATACAATCTCTTAGTCGACT	
GNOM-TG1-gRT#+	ACTACACTTGTCAACAGAGC	For cloning 4 sgRNA expression cassettes targeting GNOM
GNOM-TG1-AtU3bT#	GCTCTGTTGACAAGTGTAGTTGACCAATGTTGCTCC	
GNOM-TG2-gRT#+	TTGATGGATGATGGACCAGT	
GNOM-TG2-AtU3dT#	ACTGGTCCATCATCCATCAATGACCAATGGTGCTTTG	
GNOM-TG3-gRT#+	GTG TACTCATCAAGATGGAC	
GNOM-TG3-AtU6-1T#	GTCCATCTTGATGAGTACCAATCACTACTTCGTCT	
GNOM-TG4-gRT#+	TCAGCTCATCTACAGTCAAT	
GNOM-TG4-AtU6-29T#	ATTGACTGTAGATGAGCTGACAATCTCTTAGTCGACT	



attB2-AtU3b-F	<u>GGGGACAGCTTTCTGTACAAAGTGG</u> AATTTACTTTAAATT TTTTCTTAT	Generating p2R3z- AtU3b-tRNA-ccdB-gRNA entry clone
tRNA-AtU3b-R	ACCACTAGACCACTGGTGCTTTGTTTGACCAATGTTGCTCC CTCAGTGTT	
AtU3b-tRNA-F	TAACACTGAGGGAGCAACATTGGTCAAACAAAGCACCAGT GGTCTA	
tRNA-R	CCGTGGCAGGGTACTATTCTACCACTAGACCACTGGTGCT TTGTT	
tRNA-F	AGAATAGTACCCTGCCACGGTACAGACCCGGGTTTCGATTC CCGGCT	
ccdB-tRNA-R	TGAATCGGCCACTTCAGAGGTCTCTTGACCAGCCGGGAA TCGAACCCGGG	
tRNA-ccdB-F	CCCGGGTTCGATTCGGGCTGGTGCAAGAGACCTCTGAAG TGGCCGATTCA	
ccdB-sgRNA-R	AACTTGCTATTTCTAGCTCTAAAACCGAGACCCACGCTCAC CCGCCGCGC	
ccdB-sgRNA-F	GCGCGGCGGGTGAGCGTGGGTCTCGGTTTTAGAGCTAGA AATAGCAAGTT	
attB3-sgRNA-R	<u>GGGGACA</u> ACTTTGTATAATAAAGTTGAAAAAAAAAAGCAC CGACTCGGTGCCA	
BSAI-PLT2-TG1-F	TGCAT <u>GTGAAGAGTGAATGTGAGG</u>	
BSAI-PLT2-TG1-R	AAACCCTCACATTCACTCTTCACA	
CAS9-RFP-F	CGTATCGACCTTTCCAGCTTGGTGGTGATATGAGCGAGC TGATTAAGGA	For making p221z-Cas9- tagRFP entry clone
NLS-RFP-R	TCCGGCCTTTTGGTGGCAGCAGGACGCTTCTTGTGCCCC AGTTTGCTAG	

Underlined sequences indicate Gateway adaptors. Sequence in red represent the target sequence in the gene.

**Supplementary Table 3 Constructs list in this study.**

expression vector name	1st BOX	2nd BOX	3rd BOX	destination vector
35S:XVE>>CAS9p-PLT2-AtU3b-sgRNA1	p1R4-35S-XVE	p221z-CAS9p-T35S	p2R3z-PLT2-AtU3b-sgRNA1	pBm43GW
35S:XVE>>CAS9p-PLT2-AtU3d-sgRNA1	p1R4-35S-XVE	p221z-CAS9p-T35S	p2R3z-PLT2-AtU3d-sgRNA1	pBm43GW
35S:XVE>>CAS9p-PLT2-AtU6-1-sgRNA1	p1R4-35S-XVE	p221z-CAS9p-T35S	p2R3z-PLT2-AtU6-1-sgRNA1	pBm43GW
35S:XVE>>CAS9p-PLT2-AtU6-29-sgRNA1	p1R4-35S-XVE	p221z-CAS9p-T35S	p2R3z-PLT2-AtU6-29-sgRNA1	pBm43GW
35S:XVE>>CAS9p-PLT2-AtU3b-tRNA-sgRNA1	p1R4-35S-XVE	p221z-CAS9p-T35S	p2R3z-PLT2-AtU3b-tRNA-sgRNA1	pFRm43GW
35S:XVE>>CAS9p-PLT2-sgRNA1-4	p1R4-35S-XVE	p221z-CAS9p-T35S	p2R3z-PLT2-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pBm43GW
35S:XVE>>dCAS9p-PLT2-sgRNA1-4	p1R4-35S-XVE	p221z-dCAS9p-T35S	p2R3z-PLT2-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pBm43GW
35S:XVE>>CAS9p-tagRFP-PLT2-AtU3b-sgRNA1	p1R4-35S-XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-PLT2-AtU3b-sgRNA1	pBm43GW
35S:XVE>>AtMIR390-PLT2-1-nosT2	p1R4-35S-XVE	p221z-AtMIR390-PLT2-1	nosT2	pFRm43GW
35S:XVE>>AtMIR390-PLT2-2-nosT2	p1R4-35S-XVE	p221z-AtMIR390-PLT2-2	nosT2	pFRm43GW
pWOX5:XVE>>AtMIR390-PLT2-1-nosT2	p1R4-pWOX5:XVE	p221z-AtMIR390-PLT2-1	nosT2	pFRm43GW
pWER:XVE>>CAS9p-tagRFP-PLT2-sgRNA1-4	p1R4-pWER:XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-PLT2-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pBm43GW
pWOX5:XVE>>CAS9p-tagRFP-PLT2-sgRNA1-4	p1R4-pWOX5:XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-PLT2-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pBm43GW
pSCR:XVE>>CAS9p-tagRFP-PLT2-sgRNA1-4	p1R4-pSCR:XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-PLT2-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pBm43GW
pWOL:XVE>>CAS9p-tagRFP-PLT2-sgRNA1-4	p1R4-pWOL:XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-PLT2-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pBm43GW
pWER:XVE>>CAS9p-taRFP-RBR-sRNA1-4	p1R4-pWER:XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-RBR-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pFRm43GW
pWOX5:XVE>>CAS9p-taRFP-RBR-sRNA1-4	p1R4-pWOX5:XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-RBR-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pFRm43GW
pSCR:XVE>>CAS9p-taRFP-RBR-sRNA1-4	p1R4-pSCR:XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-RBR-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pFRm43GW
pWOL:XVE>>CAS9p-taRFP-RBR-sRNA1-4	p1R4-pWOL:XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-RBR-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pFRm43GW
35S:XVE>>CAS9p-RBR-sgRNA1-4	p1R4-35S-XVE	p221z-CAS9p-T35S	p2R3z-RBR-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pFRm43GW
pWER:XVE>>CAS9p-tagRFP-AtU3b-YFP-sgRNA	p1R4-pWER:XVE	p221z-CAS9p-tagRFP-T35S	2R3z-VEN-AtU3b-sgRNA	pFRm43GW
pWOX5:XVE>>CAS9p-tagRFP-AtU3b-YFP-sgRNA	p1R4-pWOX5:XVE	p221z-CAS9p-tagRFP-T35S	2R3z-VEN-AtU3b-sgRNA	pFRm43GW
pWOL:XVE>>CAS9p-tagRFP-GNOM-sgRNA1-4	p1R4-pWOL:XVE	p221z-CAS9p-tagRFP-T35S	p2R3z-GNOM-AtU3b-sgRNA1+AtU3d-sgRNA2+AtU6-1-sgRNA3+AtU6-29-sgRNA4	pFRm43GW