SUPPLEMENTARY FIGURES

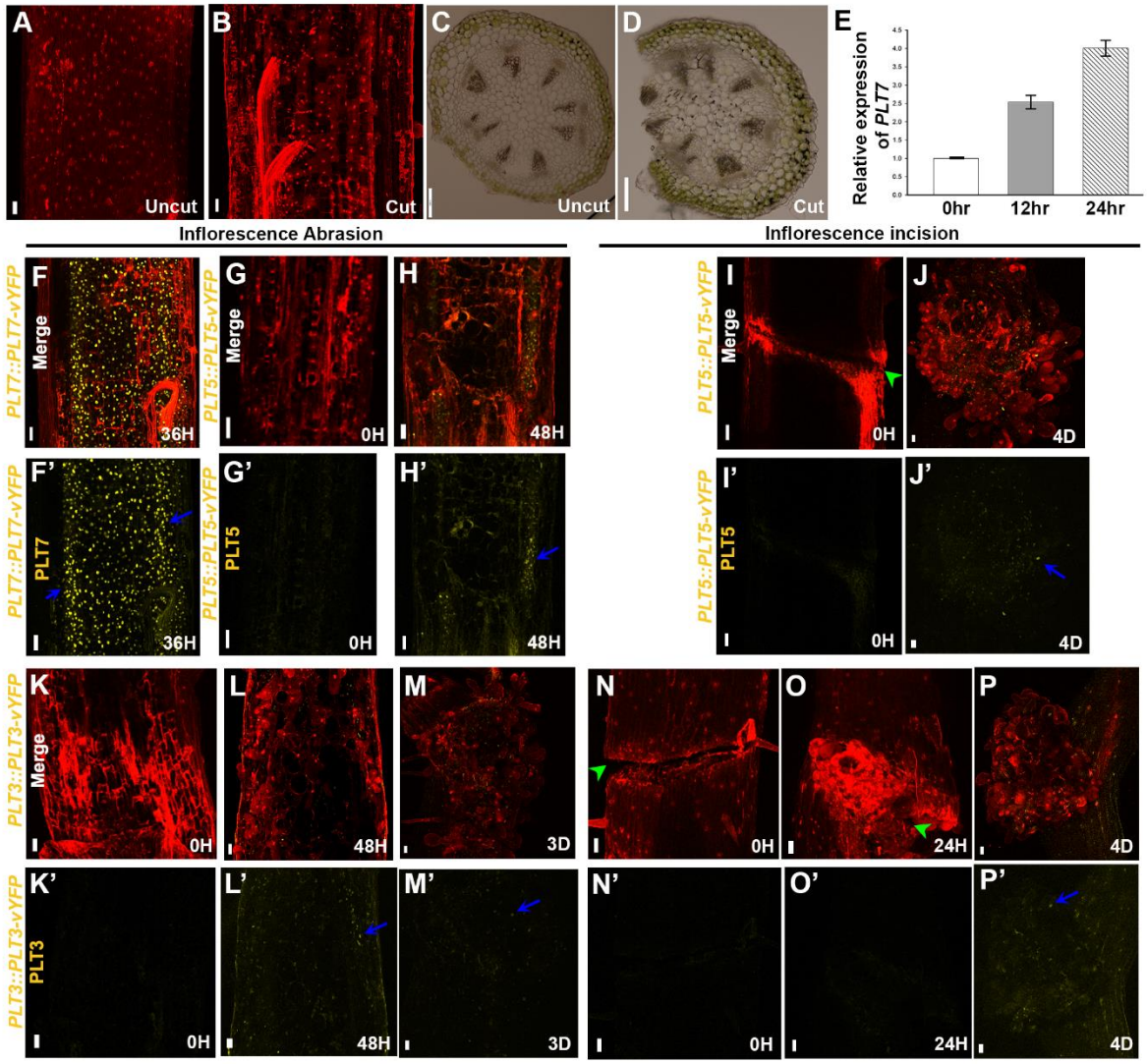

Figure S1: Dynamic expression of PLT in response to inflorescence stem injury

Inflorescence stem abrasion causes damage to epidermal and vascular tissues: (A, C) Undamaged inflorescence stem. (B, D) Sections revealing damaged epidermis and sub-epidermal layers including vascular tissue post inflorescence stem abrasion. A and B represent longitudinal sections. C and D represent transverse sections. Red colour in A, B is propidium iodide staining.

PLT7 transcript level in wild type upon partial incision in inflorescence stem: (E) Injured inflorescence stem segment encompassing the narrow domain on either side of partial slit were

collected at 0 h, 12 h and 24 h. Expression levels are normalized to *ACTIN2*. Error bar represents s.e.m. from three independent biological replicates.

PLT proteins show dynamic expression in growing aerial organs during wound healing: (F, F') Expression of *PLT7::PLT7-vYFP* in response to inflorescence stem abrasion. Note the expression of *PLT7::PLT7-vYFP* in sub-epidermal tissues and near vascular tissue (blue arrow). (G-J') Expression of *PLT5::PLT5-vYFP* during natural regeneration. Response to inflorescence stem abrasion (G-H') and inflorescence stem partial incision (green arrowhead) (I-J'). Note the increase in expression of *PLT5::PLT5-vYFP* in wounded vascular tissue in H' (blue arrow). (J') Weak expression of *PLT5::PLT5-vYFP* in callus formed in response to injury. (K-P') Expression of *PLT3::PLT3-vYFP* during natural regeneration. Response to inflorescence stem abrasion (K-M, K'-M') and inflorescence stem partial incision (N-P,N'-P'). Weak expression of *PLT3::PLT3-vYFP* is observed in sub-epidermal tissues (L') and in the callus formed in response to wounding (M' and P').

(F'-J' and K'-P'): maximum intensity projection of z stack in YFP channel corresponding to (F-J and K-P). Red colour represents propidium iodide staining. Green arrowheads: partial incision in inflorescence stem. Blue arrows: Expression of *PLT* in response to wounding. Scale bar: 50 µm except in C and D where scale bars represent 1 mm. Brightness of YFP channel has been increased in H', J', L', M' and P' for visibility. The panels (F-P) represent different samples at each time point.

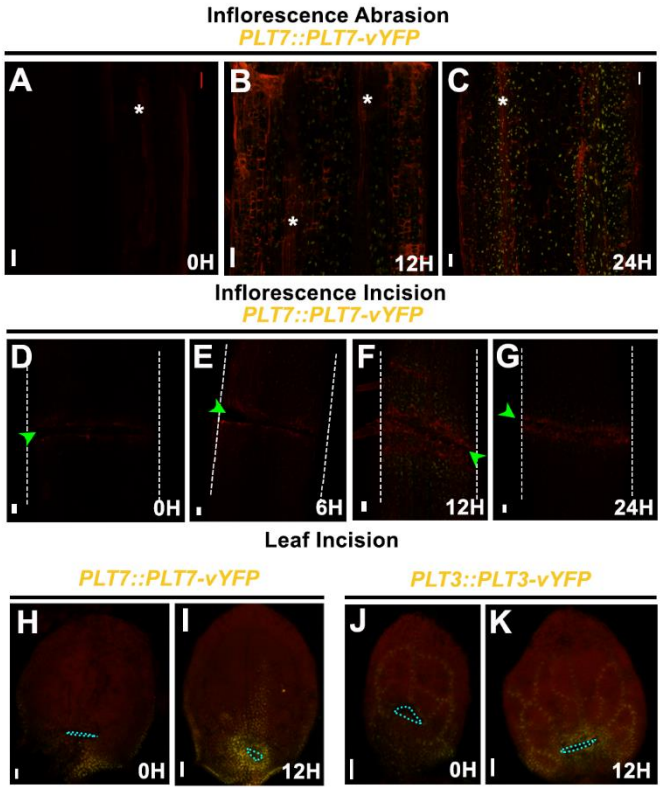

Figure S2: *PLT3, PLT5* and *PLT7* genes are locally induced after mechanical injury

(A-G) *PLT7::PLT7-vYFP* expression (yellow) post abrasion (A-C) and partial incision (green arrowhead) (D-G) in growing inflorescence stems. White asterisks: vascular tissues exposed by damage to epidermal and sub-epidermal layers following local abrasion. White dashed line: Inflorescence stem outline. (H-K) Upregulation of *PLT7::PLT7-vYFP* (H, I) and *PLT3::PLT3-vYFP* (J, K) (yellow) near wound site following leaf incision (blue dotted area: incision site). The panels represent average intensity projections of merged panels in Fig. 1 and each panel represent different samples at each time point. Red signal is propidium iodide staining in (A-G) and chlorophyll autofluoroscence in (H-K). Scale bars:50 μm.

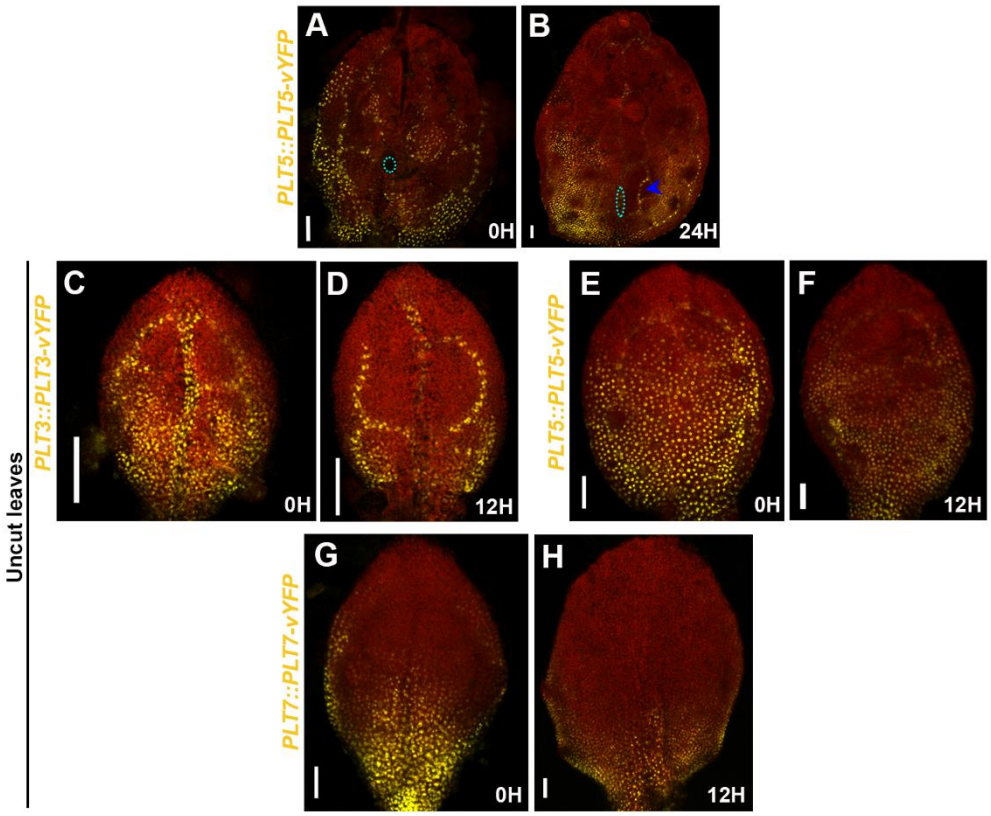

Figure S3: PLT expression in injured and undamaged leaves

(A, B) *PLT5::PLT5-vYFP* expression in adjacent vascular strand (blue arrowhead) post incision (B).

(C-H) Expression of *PLT3::PLT3-vYFP*(C, D), *PLT5::PLT5-vYFP* (E, F), *PLT7::PLT7-vYFP* (G, H), in wild type undamaged leaves.

Red colour represents chlorophyll autofluorescence. B represents a subset of z stack. Brightness and contrast have been adjusted in chlorophyll autofluorescence channel for clarity of injured part. Blue dotted area: site of incision. Scale bars: 50 μm.

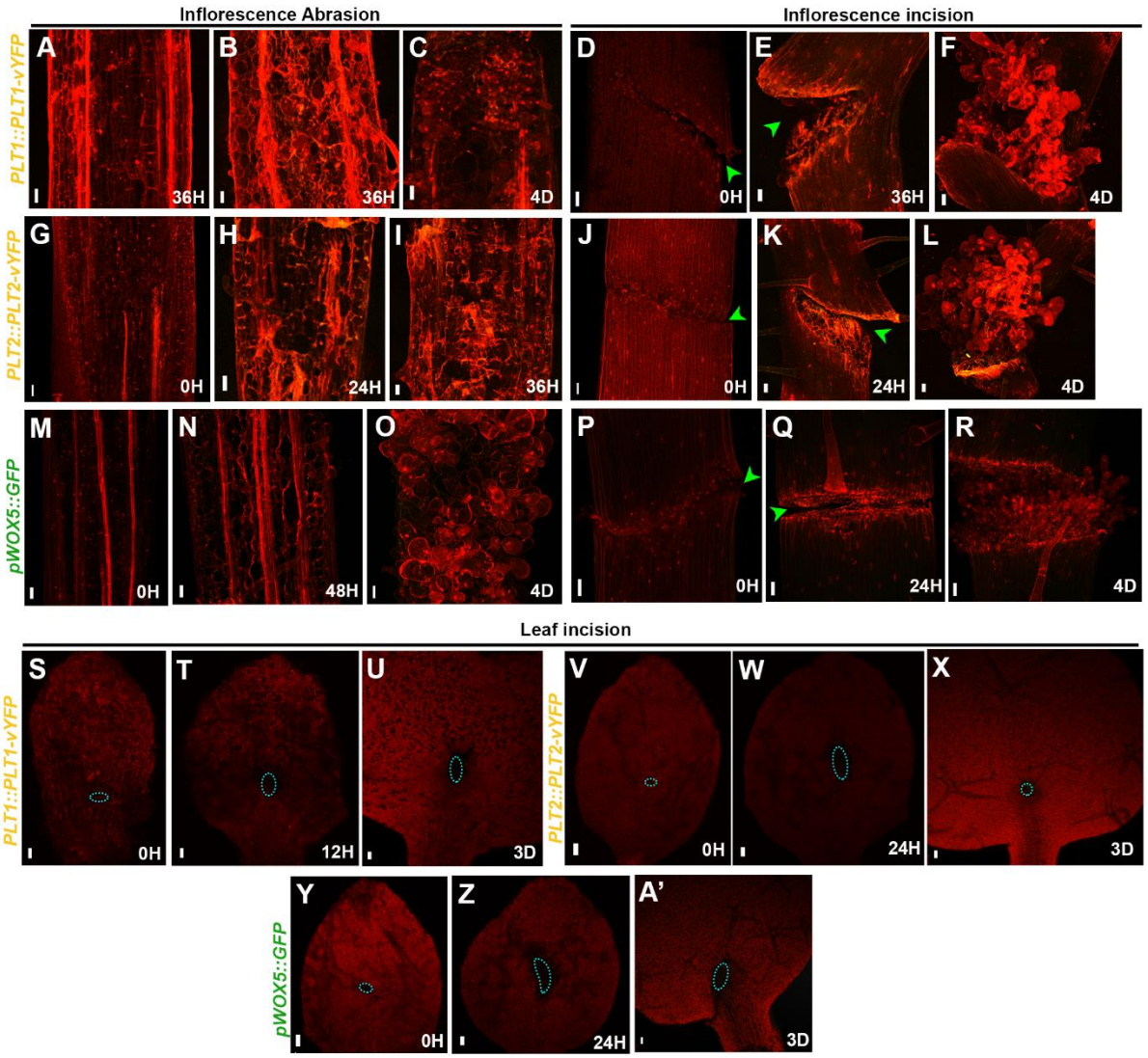

Figure S4: Absence of root stem cell regulators during wound repair in aerial organs

(A-A') Absence of *PLT1::PLT1-vYFP* (A-F, S-U), *PLT2::PLT2-vYFP* (G-L, V-X) and *pWOX5::GFP* (M-R, Y-A') following injury in growing aerial organs. Red colour in (S-A') represent chlorophyll autofluorescence and propidium iodide staining in the rest. Green arrowheads: partial incision in inflorescence stems. Blue dotted area: incision sites. Scale bars: 50 μm. Brightness and contrast have been adjusted in propidium iodide channel for clarity.

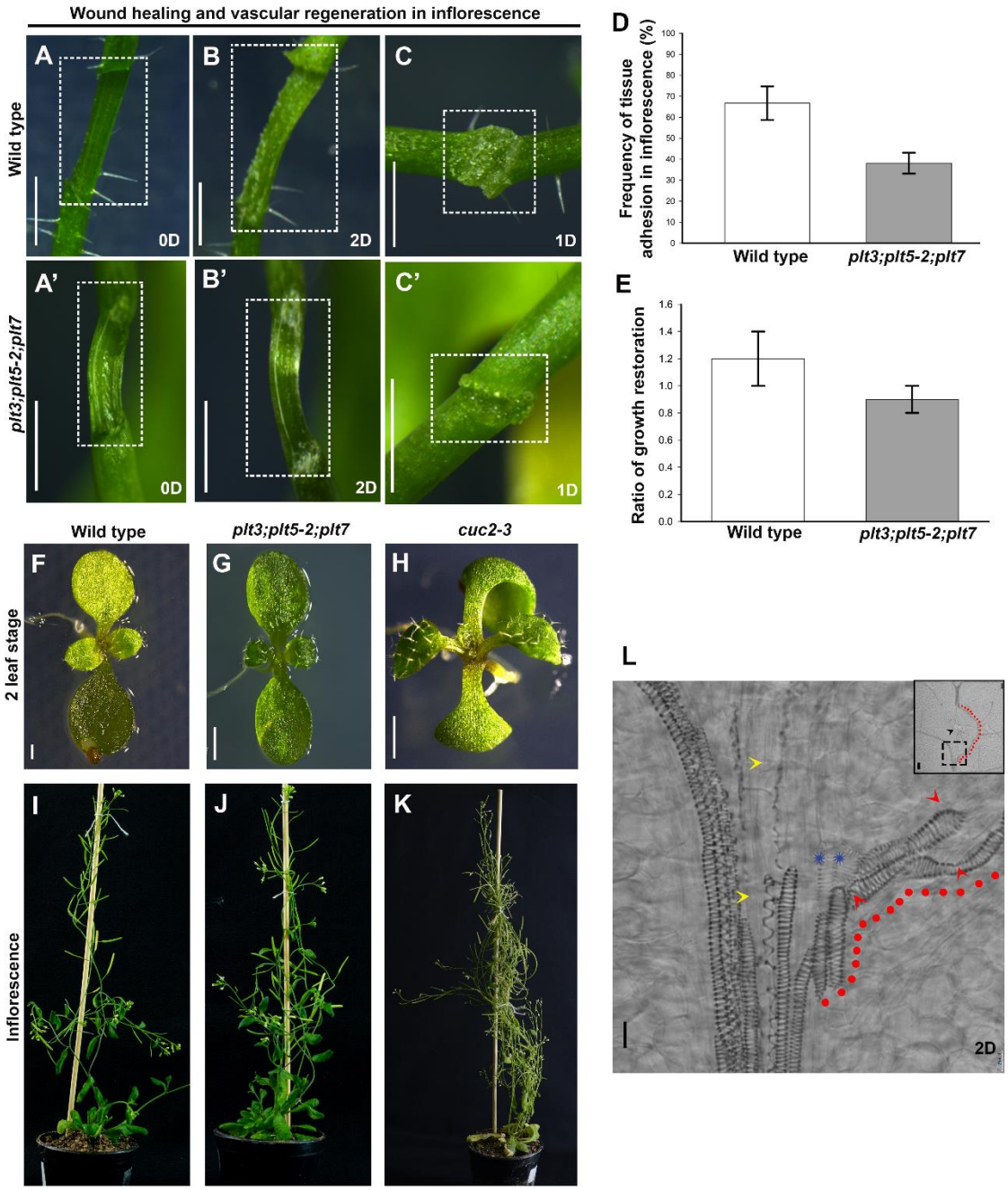

Figure S5: PLT activate innate regenerative responses to injuries in aerial organs growing in normal developmental context

(A, A') Inflorescence stem abrasion in wild type (A) and *plt3;plt5-2;plt7* (B). (B, B') Wild type inflorescence stem with cell proliferation (B) while *plt3;plt5-2;plt7* (B') inflorescence stem failed to show any proliferation. (C, C', D) More callus formation in wild type (C) 24 h following partial incision on inflorescence stem leading to increased frequency of tissue adhesion (D) in wild type as compared to *plt3;plt5-2;plt7* (C'). Dotted rectangle: area of

inflorescence stem damage. (E) Graph representing growth restoration in wild type and *plt3;plt5-2;plt7* post partial incision in inflorescence stem.

(F-K) Mutants do not display defect in the normal growth of leaves and inflorescence stems as compared to wild type.

(L) Zoomed in image shows lower cut end of midvein, two days post leaf incision. Yellow arrowheads mark degenerating vascular strands at lower cut end of midvein. Blue star: initiation of procambium differentiation into vascular cells. Red arrowheads: differentiated xylem vessel elements formed in response to injury. Red dots indicate regenerating vascular stand. Inset shows lower magnification image with black arrow marking site of leaf incision. Area enclosed in dashed line within inset is enlarged in (L).

Scale bar:1 mm in all panels except L (Scale bar: 50 μm). Error bars represent s.e.m. in all cases.

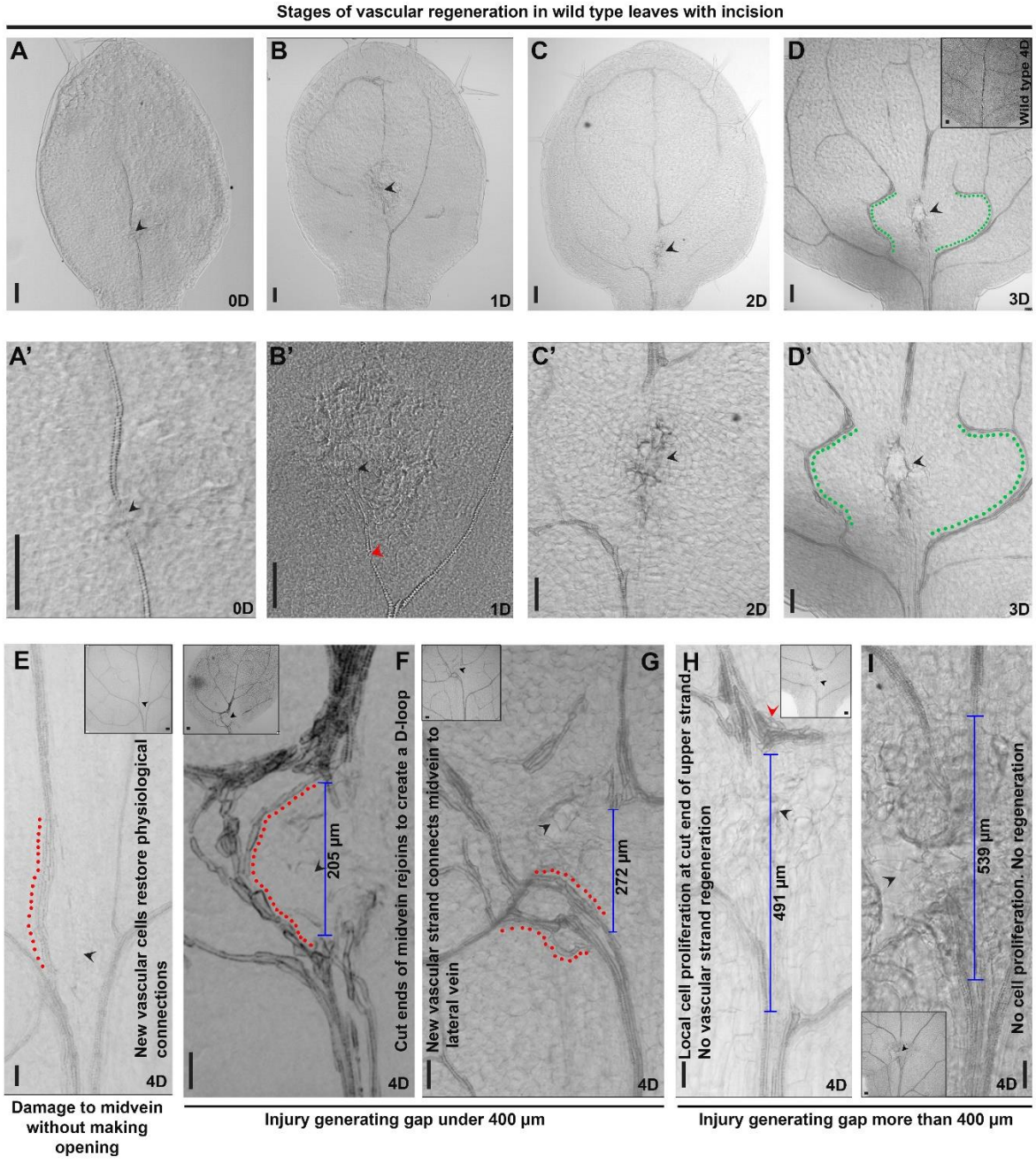

Figure S6: Response to midvein injury in leaf is dependent on the extent of tissue damage

(A-D') Stages of vascular regeneration in wild type leaves with incision: (A, A') Incision (black arrow) in midvein of 5 dpg old wild type leaf. Note that only midvein is differentiated at this stage. (B, B') Wild type leaf with incision on midvein 1 day post injury. Red arrow head: degenerating vascular strand. (C, C') Wild type leaf with incision on midvein 2 days post injury. (D, D') Wild type leaf with incision on the midvein 3 days post injury. New vascular cells form between lateral veins creating a venation pattern (green dots) which does not occur in uninjured wild type leaf (inset). (A'-D') Higher magnification images of panels

corresponding to (A-D).

(E-I) Responses to midvein injury in growing leaf. (E) Regeneration of new vascular cells (red dotted line) restore physiological connection in midvein. (F) Regenerating vascular strands (red dotted lines) rejoins disconnected ends of midvein by creating a D shaped loop (G) Regenerating vascular strands rejoins lower cut end of midvein to lateral vein. (H) Local cell proliferation (red arrow) at the cut end of upper strand but no regeneration of vascular strands. (I) No vascular cell proliferation or regeneration due to extensive area of damage creating opening in the leaf. Insets: Lower magnification images showing site of incision. Black arrowheads: Site of incision.

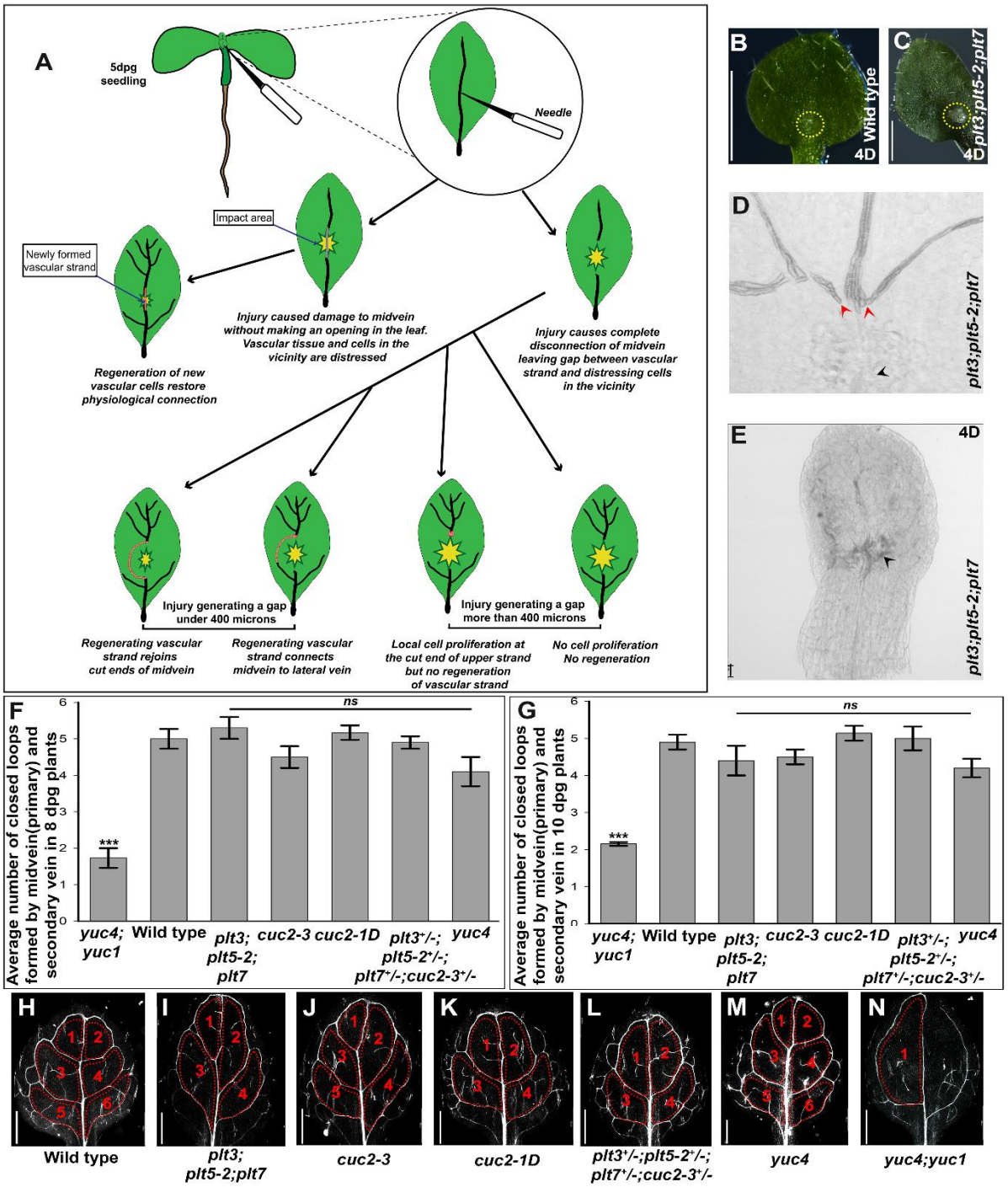

Figure S7: Normal development of vein loops in wild type and mutants

(A) Schematic representation showing vascular regeneration in response to injuries of varying sizes in the midvein of growing leaf.

(B-D) No local cell proliferation was observed on wild type leaf surface (B). Proliferation in epidermis (C) and vascular strand (D) (red arrowhead) of *plt3;plt5-2;plt7* following leaf incision (site of incision marked by yellow dotted circle/ black arrowhead).

(E) Following incision many of the *plt3;plt5-2;plt7* mutant leaves display stunted growth and slower development. Black arrowhead: site of incision.

(F, G) Number of vein loops formed by primary and secondary veins showing continuity of formation of midvein and lateral veins during normal development of first pair of wild type and mutant leaves (collected from 8 dpg and 10 dpg plants). (8 dpg samples: Kruskal–Wallis $\chi 2$ test; *P* value: *plt3;plt5-2;plt7*=0.7; *cuc2-3*=0.3; *cuc2-1D*=0.6; *plt3*$^{+}$*/-;plt5-2*$^{+}$*/-;plt7*$^{+}$*/-;cuc2-3*$^{+}$*/-*=0.8; *yuc4=0.06; yuc4;yuc1*=2x10^{-16}) (10 dpg samples: Kruskal–Wallis $\chi 2$ test*; P* value: *plt3;plt5-2;plt7*=0.2; *cuc2-3*=0.3; *cuc2-1D*=0.5; *plt3*$^{+}$*/-;plt5-2*$^{+}$*/-;plt7*$^{+}$*/-;cuc2-3*$^{+}$*/-*=0.8; *yuc4*=0.35; *yuc4;yuc1*=3.5x10^{-14}).

(H-N) Venation pattern in leaves of wild type and mutants: Mutants (except negative control-*yuc4;yuc1*) does not show significant change in formation of closed vein loops compared with wild type leaves. Red dotted lines and numbers mark closed vein loops formed by primary vein (midvein) and secondary vein (lateral vein).

Error bars represent s.e.m. in all cases.

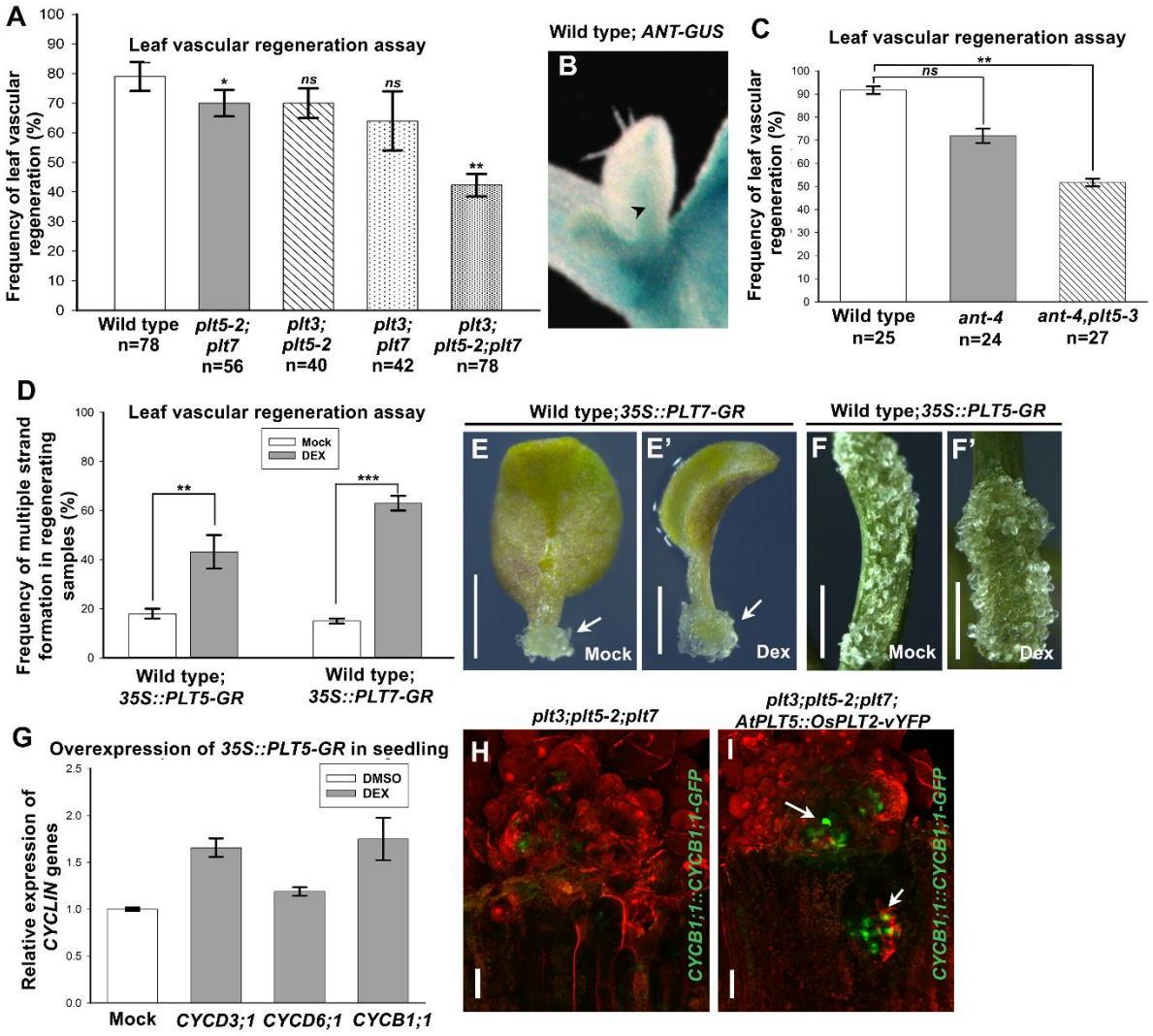

Figure S8: PLT5 and PLT7 are sufficient to promote multiple strand formation during vascular regeneration and wound repair.

(A) Frequency of leaf vascular regeneration in wild type, *plt* double mutants and *plt3;plt5-2;plt7* triple mutants (Pearson's $\chi 2$ test ;**P* =0.025;***P* =0.008;ns, *P* >0.05).

(B) Expression of *AINTEGUMENTA* in leaf vasculature (black arrow).

(C) Frequency of leaf vascular regeneration in wild type, *ant4* mutant and *ant4;plt5-3* double mutant (Pearson's $\chi 2$ test; ns, *P* >0.05;***P* =0.004).

(D) Increased multiple strand formation upon overexpression of *35S::PLT5-GR* and *35S::PLT7-GR* during vascular regeneration in response to midvein incision (Pearson's $\chi 2$ test ;***P* =0.007; ****P* =1.2x10^{-5}). (E, E') Increased callus formation (white arrow) from cut end of leaf on ectopic induction of *35S::PLT7-GR* (E') as compared to control (E). (F, F') Increased callus formation on the surface of inflorescence stem following abrasion and induction of *35S::PLT5-GR* (F') as compared to control (F). Error bars in A, C and D represent s.e.m.

(G) Expression of *CYCLIN* genes in response to overexpression of *35S::PLT5-GR* in growing

seedlings. Expression levels are normalized to *ACTIN2*. Error bar represents s.e.m. from three independent biological replicates.

(H, I) *plt3;plt5-2;plt7* (H) barely shows any cell proliferation marked by cell cycle progression marker *CYCB1;1::CYCB1;1-GFP* as compared to strong expression detected in clusters (white arrow) of actively dividing cells forming callus in response to inflorescence stem abrasion in *plt3;plt5-2;plt7;AtPLT5::OsPLT2-vYFP* (I). Confocal imaging was performed only for GFP excitation and emission detection.

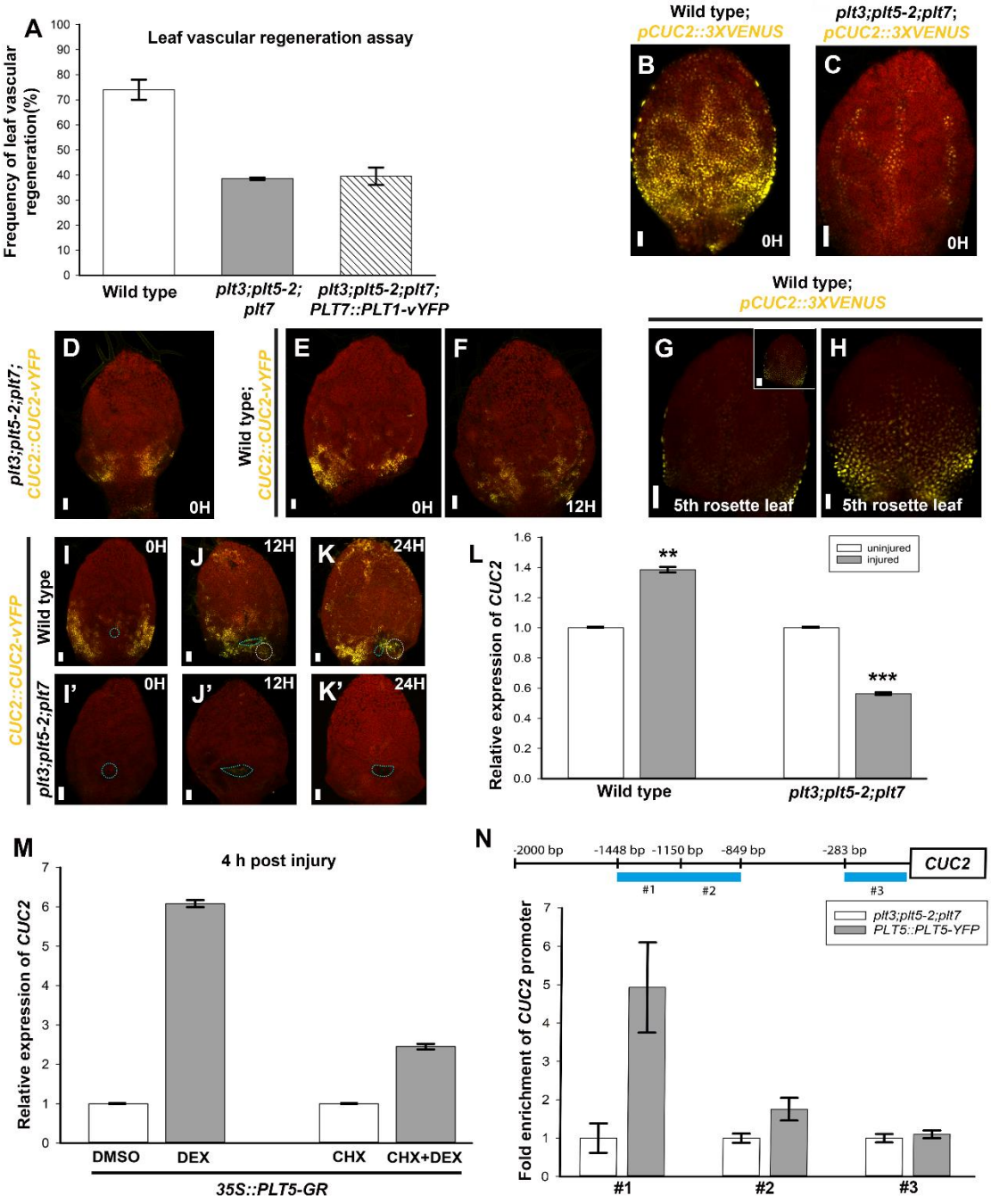

Figure S9: PLT directly activates CUC2 during wound response

(A) Leaf vascular regeneration in wild type, *plt3;plt5-2;plt7* and *plt3;plt5-2;plt7;PLT7::PLT1-vYFP*

(B-H) Expression of *CUC2* in undamaged leaves. Expression of *pCUC2::3XVENUS* (B,C) and *CUC2::CUC2-vYFP* (D-F) in undamaged leaves. (G) Single optical section showing expression of *pCUC2::3XVENUS* in the leaf margin of fifth rosette leaf. Inset in (G) represents stacked image of the same leaf. (H) *pCUC2::3XVENUS* expression is absent from the hydathode and higher in the leaf sinus as reported previously (Nikovics *et al.*, 2006;

Bilsborough *et al.*, 2011). Except (G) and (H) (5th rosette leaves), all other panels present leaves belonging to 1st pair of rosette leaves.

(I, I') *plt3;plt5-2;plt7* shows reduced expression of *CUC2::CUC2-vYFP* as compared to wild type.

(J-K') Upon incision wild type (J,K) shows expanded domain of expression of *CUC2::CUC2-vYFP* unlike *plt3;plt5-2;plt7* (J',K'). White dotted circle marks upregulation of YFP expression near wounded area. Blue dotted line marks incision.

(L) Upregulation of *CUC2* transcript in injured wild type leaf at 12 h post injury as compared to control uninjured wild type leaves. Downregulation of *CUC2* transcript in injured *plt3;plt5-2;plt7* leaves as compared to control uninjured *plt3;plt5-2;plt7* leaves. (Welch's two-sample t-test; $^{**}P$ =0.002;$^{***}P$ =0.0004)

(M) Transcript level of *CUC2* upon induction of PLT5 with DEX treatment and with cycloheximide treatment.

Expression levels in (L) and (M) are normalized to *ACTIN2*. Error bar represents s.e.m. from three independent biological replicates

(N) ChIP-qPCR Analysis: ChIP-qPCR experiment in callus tissues shows direct binding of PLT5 fusion protein to the *CUC2* promoter. The results are shown as fold enrichment relative to *plt3;plt5-2;plt7* loss of function mutant. A strong binding of PLT5 is noticed at the fragment #1 (-1150 to -1448 bp) followed by a weak binding at #2 (-849 to -1149 bp) and no significant binding at the fragment #3 (-1 to -283 bp) of the upstream sequence of *CUC2*. Error bars show the standard error of the ChIP-qPCR reactions performed in triplicates.

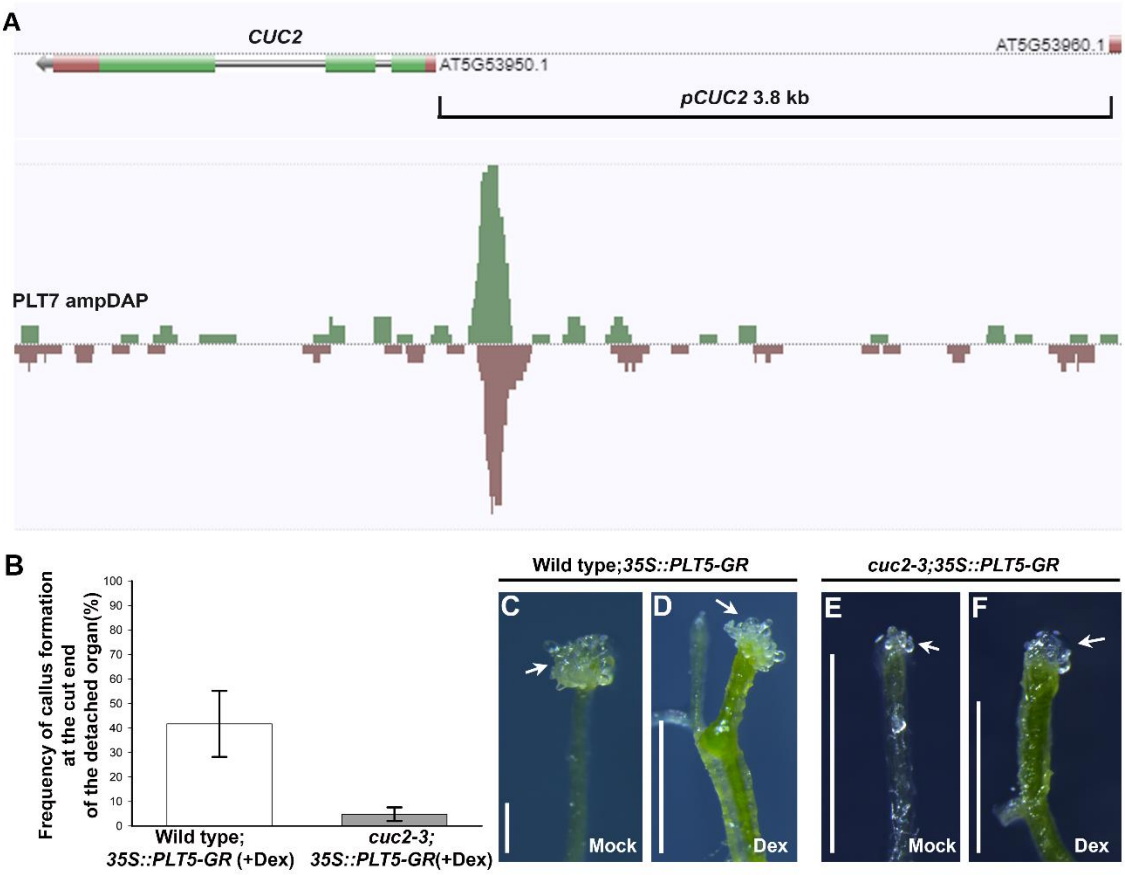

Figure S10: PLT acts through CUC2 during wound repair

(A) PLT7 binds the *CUC2* promoter (http://neomorph.salk.edu/). Indicated region shows *pCUC2*, which was used in the luciferase reporter assay.

(B) Frequency refers to the number of excised organs showing callus formation at the cut end. In addition to frequency, the extent of callus formation is lesser in *cuc2-3;35S::PLT5-GR*.

(C,D) Wild type;*35S::PLT5-GR* upon continuous DEX induction (n=12/15) (D) following excision shows increased extent of callus formation unlike in mock treated control (n=9/10) (C) at the detached end of root.

(E,F) *cuc2-3;35S::PLT5-GR* upon continuous DEX induction (n=15/20) (F) following excision shows no increase in extent of callus formation at the detached end of root as compared to mock treated control (n=16/20) (E).

Arrow: Callus formation. Scalebars:1 mm.

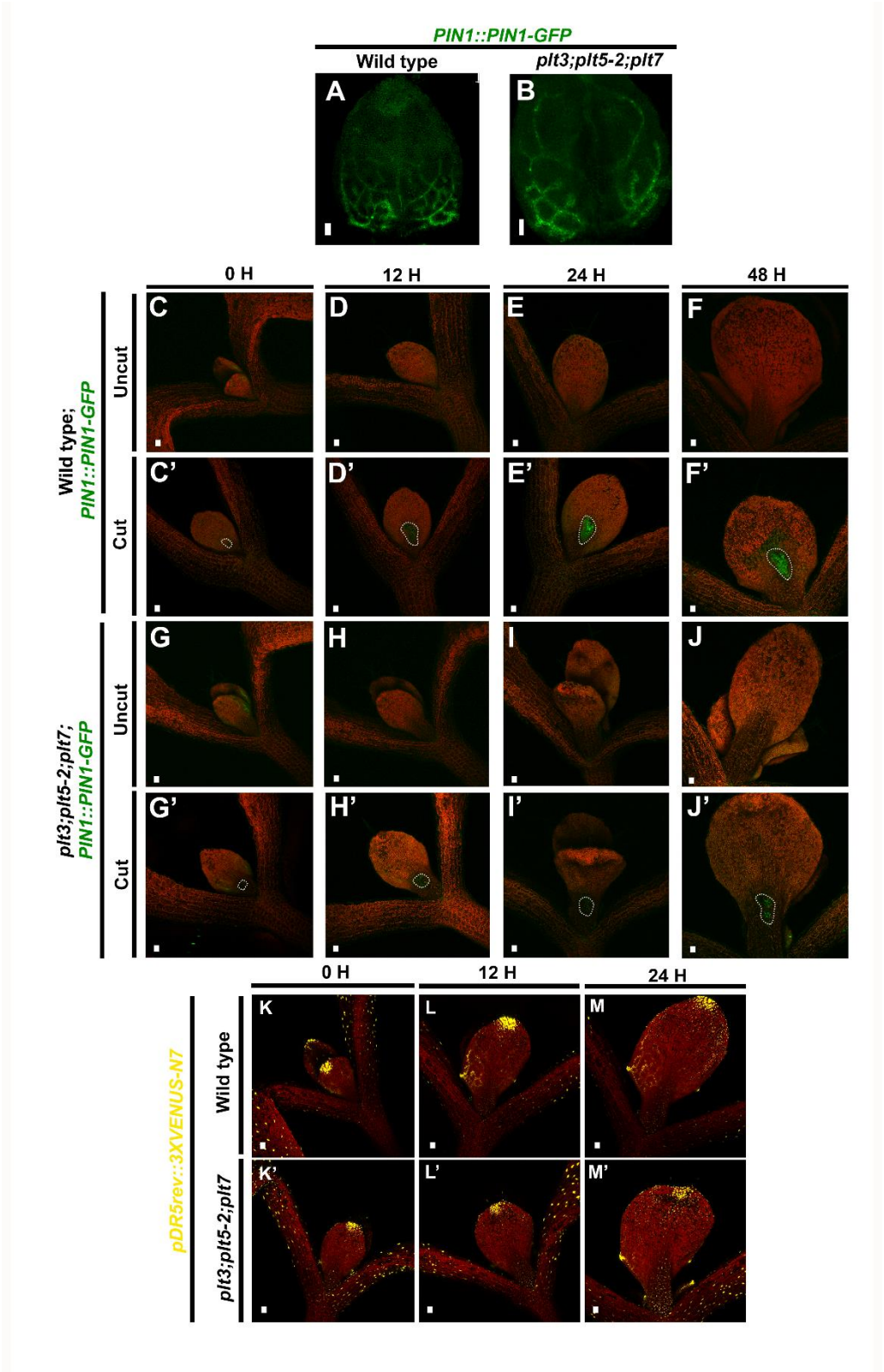

Figure S11: PIN1 expression and auxin response are not defective in *plt* mutant during normal development

(A,B) *PIN1::PIN1-GFP* expression in undamaged leaves of wild type (A) and *plt3;plt5-2;plt7* (B). PIN1 expression is visible in the basal part of the leaves in both wild type and *plt3;plt5-2;plt7*.

(C-J') Confocal time lapse images showing expression of *PIN1::PIN1-GFP* in wild type (C-F') and *plt3;plt5-2;plt7* (G-J'). (C-F) and (G-J) represent uninjured leaves while the remaining represent injured leaves in which injured areas are marked by white dotted lines.

(K-M') Confocal time lapse images showing expression of *pDR5rev::3XVENUS-N7* in wild type (K-M) and *plt3;plt5-2;plt7* (K'-M') uninjured leaves.

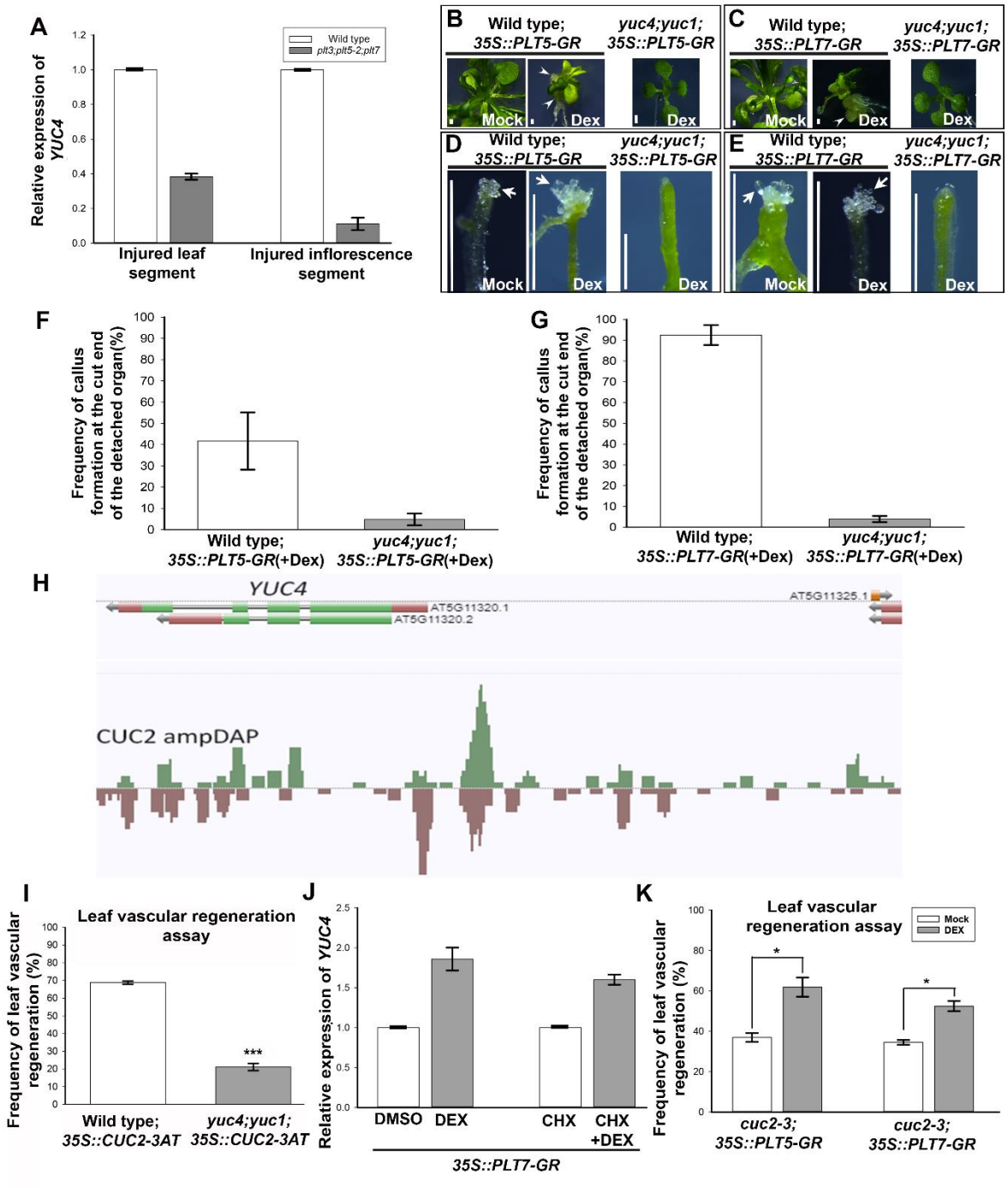

Figure S12: PLT acts though YUC4 during reprogramming and wound repair

(A) *YUC4* transcript level in injured and uninjured leaf and inflorescence stem segments of wild type and *plt3;plt5-2;plt7* mutant. Expression levels in A is normalized to *ACTIN2*. Error bar represents s.e.m. from three independent biological replicates.

(B) Growing seedlings of Wild type;*35S::PLT5-GR* upon DEX induction shows callus formation (arrowheads) from shoot and root leading to stunted growth of the plant, unlike mock treated control, which does not show any ectopic phenotypes. However *yuc4;yuc1;35S::PLT5-GR* does not show any cellular reprogramming even upon DEX induction.

(C) Growing seedlings of Wild type;*35S::PLT7-GR* upon DEX induction shows callus formation (arrowhead) from hypocotyl and root leading to stunted growth of the plant, unlike mock treated control, which does not show any ectopic phenotypes. However *yuc4;yuc1;35S::PLT7-GR* does not show any cellular reprogramming even upon DEX induction.

(D) Wild type;*35S::PLT5-GR* upon DEX induction (n=15/20) shows increased extent of callus formation unlike in mock treated control of detached organ (n=10/13). However *yuc4;yuc1;35S::PLT5-GR* (n=20/20) shows barely any callus formation upon DEX induction.

(E) Wild type;*35S::PLT7-GR* upon DEX induction (n=9/10) shows increased extent of callus formation unlike in mock treated control of detached organ (n=7/11). However *yuc4;yuc1;35S::PLT7-GR* (n=14/15) rarely shows callus formation upon DEX induction.

(F,G) Frequency refers to the number of excised organs showing callus formation at the cut end. In addition to frequency, the extent of callus formation at the wounded end of detached organ was extremely reduced in *yuc4;yuc1* as compared to wild type upon DEX induction of *35S::PLT5-GR* (F) and *35S::PLT7-GR* (G)

(H) CUC2 binds the *YUC4* promoter as shown by DAP-seq analysis (http://neomorph.salk.edu/).

(I) Frequency of leaf vascular regeneration in wild type;*35S::CUC2-3AT* and *yuc4;yuc1;35S::CUC2-3AT* (***P =2x10^{-6}).

(J) Transcript level of *YUC4* upon induction of *35S::PLT7-GR* with DEX treatment and with cycloheximide treatment at 4 h post injury. Expression levels are normalized to *ACTIN2*. Error bar represents s.e.m. from three independent biological replicates.

(K) Frequency of leaf vascular regeneration upon overexpression of *35S::PLT5-GR* and *35S::PLT7-GR* in *cuc2-3* mutant (Pearson's $\chi 2$ test).

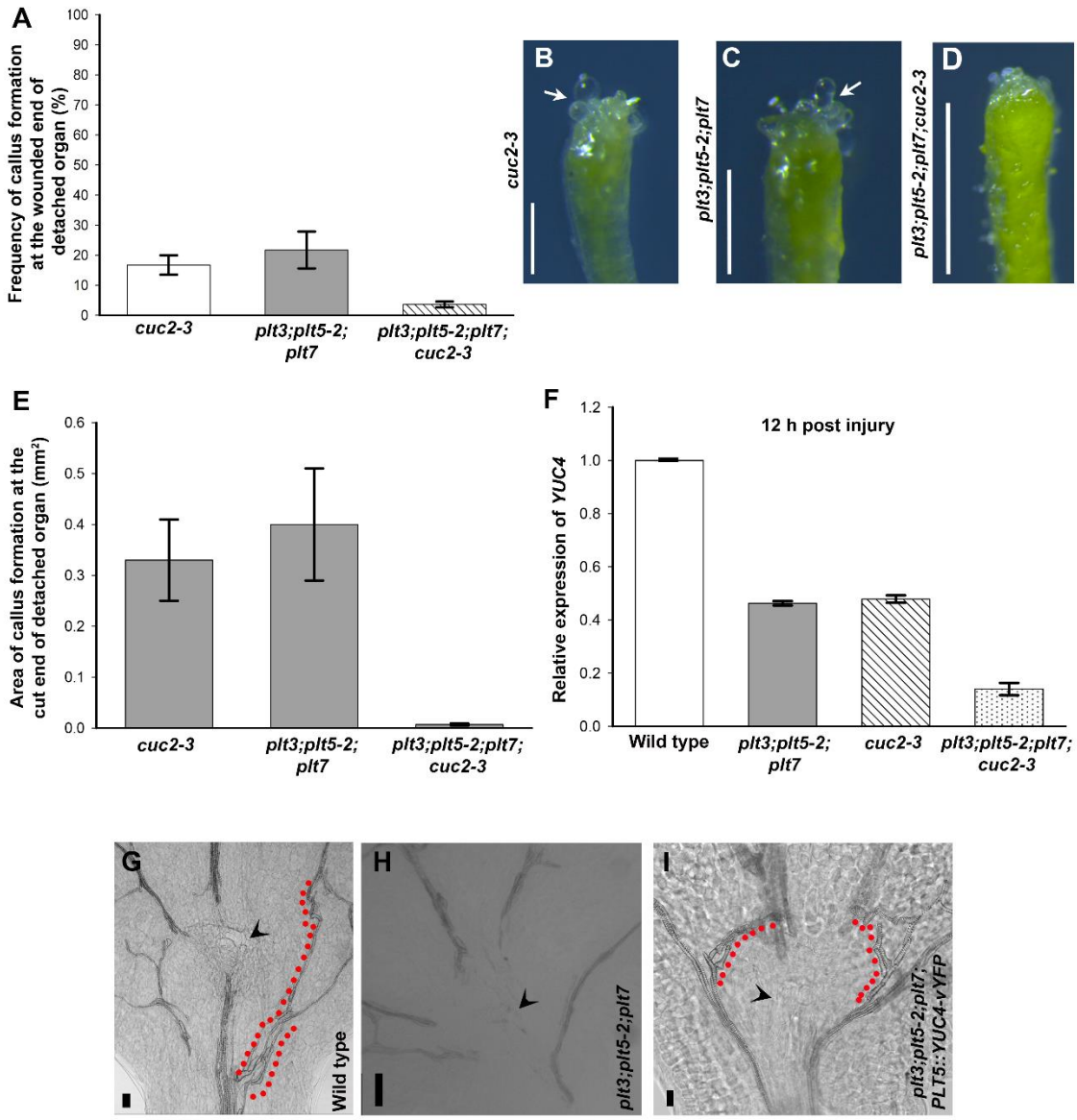

Figure S13: PLT and CUC2 regulate *YUC4* in a coherent feed forward loop

(A) Frequency refers to the number of excised organs showing callus formation at the cut end.

(B-D) In addition to frequency, extent of callus formation (white arrow) was drastically reduced in *plt3;plt5-2;plt7;cuc2-3* as compared to *cuc2-3* and *plt3;plt5-2;plt7* which showed moderate callus formation at the cut ends of detached organs.

(E) Area of callus formation at the cut end of detached organs of *cuc2-3;plt3;plt5-2;plt7* and *plt3;plt5-2;plt7;cuc2-3*.

(F) Relative expression levels of *YUC4* in wild type and mutants. Expression levels are normalized to *ACTIN2*. Error bar represents s.e.m. from three independent biological replicates.

(G-I) Vascular strand regeneration assay in wild type (G), *plt3;plt5-2;plt7* (H) and *plt3;plt5-2;plt7;PLT5::YUC4-vYFP* (I). Vascular strands fail to regenerate in *plt3;plt5-2;plt7* (H). Black arrowheads mark site of leaf incision. Red dotted lines mark regenerated vascular strands.

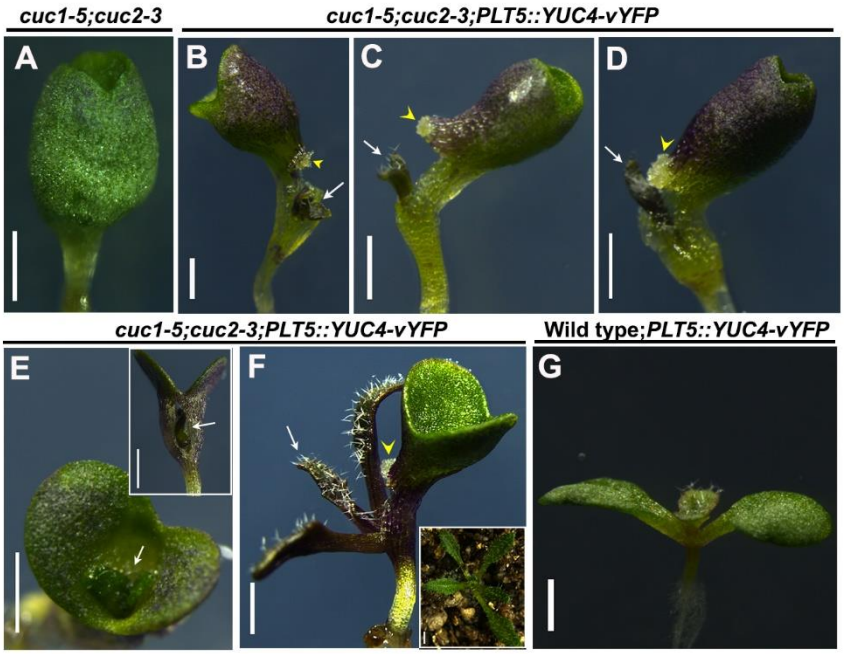

Figure S14: YUC4 rescued post embryonic development in *cuc1-5;cuc2-3* mutant

(A) Cup shaped cotyledon in *cuc1-5;cuc2-3* mutant (none out of 80 plants displaying cup shaped cotyledon produced shoot). (B-F) Reconstitution of local auxin biosynthesis gene *YUC4* in *PLT5* domain rescues post embryonic development, giving rise to fully developed leaves (marked by white arrows). Out of 48 plants with cup shaped cotyledon, 20 produced shoot from base of cotyledon. Callus formed at the base of cotyledon caused by the emergence of the shoot is marked by yellow arrowheads. (G) Wild type;*PLT5::YUC4-vYFP* showing normal shoot formation.

SUPPLEMENTARY INFORMATION

MATERIALS AND METHODS

Plasmid construction

To generate *PLT5::YUC4:vYFP* construct, 5.6kb upstream regulatory elements of *PLT5* and 1.93kb *YUC4* gene were separately amplified from genomic DNA and incorporated with *vYFP*. *plt3;plt5-2;plt7*, *cuc1-5(-/-);cuc2-3(+/-)* and *cuc2-1D* mutant plants were transformed using the construct. Similarly 1.7kb upstream regulatory element and 4.236kb *ATHB8* gene was incorporated with *vYFP* to generate the translational fusion construct *ATHB8::ATHB8-vYFP*. This construct was co-transformed with *PIN1::PIN1-GFP* into both wild type and *plt3;plt5-2;plt7* mutant to generate the double marker transgenic line. *OsPLT2 (LOC_Os06g44750.1)* was cloned under upstream regulatory elements of *Arabidopsis PLT5* gene and tagged with *vYFP*. This construct was transformed into *plt3;plt5-2;plt7*.

Decolourisation and tissue clearing for imaging vascular tissues

To visualize regenerating vascular strands, the injured leaf and inflorescence stem were carefully excised from the growing seedling 4 days post incision using Vannas straight scissors. Before proceeding for decolorization of chlorophyll, a longitudinal cut was made through the excised inflorescence stem using razor blade to expose the regenerating vascular strands. Both leaf and inflorescence stem were dehydrated and the chlorophyll was bleached by incubating the sample consecutively in 15%, 50%, 70% and 96% ethanol for 15 minutes each. Finally, the samples were incubated in absolute ethanol for 12 h. The sample was then rehydrated by transferring from 100% ethanol to 96%, 70%, 50% and finally 15% ethanol in the reverse order with 15 minutes incubation in each concentration of ethanol. Then the samples were incubated for 2-3 h in freshly prepared clearing solution consisting of 8 g chloral hydrate (Sigma-Aldrich), 1 ml 100% glycerol (Sigma-Aldrich) and 3 ml distilled water. The cleared samples were mounted on slides using the clearing solution with the abaxial surface of the leaf and the longitudinally cut surface of the inflorescence stem facing upward. Coverslip was placed carefully avoiding any bubble formation and curling of the tissues.

Sample preparation for qRT-PCR

Inflorescence stem abrasion was performed in wild type Columbia plants and *plt3;plt5-2;plt7* triple mutant. The injured part of inflorescence stem was harvested after four days and used for RNA extraction. Leaves were injured in the context of growing seedling and the entire seedling

without the root was taken for qRT-PCR. *PLT5*, *PLT7* and *CUC2* were induced using steroid inducible constructs in wild type;*35S*::*PLT5-GR*, wild type;*35S*::*PLT7-GR*, wild type;*35S*::*CUC2-GR* and *cuc1-5;cuc2-3;35S::PLT5-GR*. Prior to sample collection for RNA isolation, injured plants were transferred to MS plates containing 20 µM dexamethasone (DEX) or DMSO (Mock) (equal proportion as volume of DEX) followed by flooding the plate with liquid MS medium containing DEX or DMSO (Mock). In case of cycloheximide treatment, samples were pre-treated with 10 µM cycloheximide for 20 min (on MS medium with cycloheximide and flooded with liquid MS containing cycloheximide) followed by transfer to MS plates containing 20 µM DEX supplemented with 10 µM cycloheximide or to MS plates supplemented with DMSO and cycloheximide followed by flooding the plate with liquid MS medium of corresponding constituents. The wounded tissues were collected at 4 h or 8 h after treatment for RNA extraction.

ChIP-qPCR analysis.

600 mg fresh weight of five-day-old proliferating callus tissues derived from roots of *PLT5::PLT5-vYFP* and *plt3;plt5-2;plt7* were cross-linked in 1% formaldehyde (Sigma-Aldrich). The isolated chromatin was immunoprecipitated with anti-GFP antibody (5 µl per sample) (Clontech). After several washing steps, the protein–DNA cross-linking was reversed. Further, the DNA was cleaned using PCR Purification Kit (Qiagen).

REFERENCES FOR SUPPLEMENTARY INFORMATION

Bilsborough, G.D., Runions, A., Barkoulas, M., Jenkins, H.W., Hasson, A., Galinha, C., Laufs, P., Hay, A., Prusinkiewicz, P. and Tsiantis, M. (2011). Model for the regulation of Arabidopsis thaliana leaf margin development. *Proc. Natl. Acad. Sci. USA* **108**, 3424-3429. doi: 10.1073/PNAS.1015162108.

Kareem, A., Durgaprasad, K., Sugimoto, K., Du, Y., Pulianmackal, A.J., Trivedi, Z.B., Abhayadev, P.V., Pinon, V., Meyerowitz, E.M., Scheres, B. et al. (2015). PLETHORA genes control regeneration by a two-step mechanism.*Curr. Biol.* **25**, 1017-1030. doi: 10.1016/j.cub.2015.02.022.

Nikovics, K., Blein, T., Peaucelle, A., Ishida, T., Morin, H., Aida, M. and Laufs, P. (2006). The balance between the MIR164A and CUC2 genes controls leaf margin serration in Arabidopsis. *The Plant Cell*. **18**, 2929-2945. doi: 10.1105/TPC.106.045617.

Table S1: Synergistic interaction between PLT and CUC2 during vascular regeneration

Genotype	Frequency of leaf vascular regeneration (%)
plt3^{+}/-;*plt5-2*$^{+}$/-;*plt7*$^{+}$/-	70.52
cuc2-3^{+}/-	71.66
plt3^{+}/-;*plt5-2*$^{+}$/-;*plt7*$^{+}$/-;*cuc2-3*$^{+}$/-	36.80

Table S2. Oligonucleotide primers used for cloning and qRT PCR (5'-3')

Primer name	Forward primer	Reverse primer
qRT-PLT5	CTACTCCGGTGGACACTCGT	CGTTCTTCTTCGGAGTAGGC
qRT-PLT7	TTTCCTCGGTGATTCCTTTG	TGACGTGGATCGTAGAATGG
qRT-YUC4	TCCATAATATTAGCGACTGGGTA	CCCTTCTCTCCTTTCCATCC
pCUC2 LUCR	GGGGACAAGTTTGTACAAAAAAG CAGGCTttaattctacattttgtttgg	GGGGACCACTTTGTACAAGAA AGCTGGGTtgttttgaagaagaagataaa
ATHB8 promoter	GGGGACAACTTTGTATAGAAAAG TTGTTCGGATAAACCAATTTTCAA ATG	GGGGACTGCTTTTTTGTACAAA CTTGTCTTTGATCCTCTCCGAT CT
ATHB8 gene	GGGGACAAGTTTGTACAAAAAAG CAGGCTGTATGGGAGGAGGAAGC AATAATAGTCA	GGGGACCACTTTGTACAAGAA AGCTGGGTTTATAAAAGACCA GTTGAGGAACATGAAGC

Additional primers used in this study have been previously described (Kareem *et al*. 2015)

Table S3. Primers used for ChIP-qPCR

Primer name	Forward primer	Reverse primer
CUC2-ChIP #1	ACATTTTTGGGTGGGAAAT	AGAGAAGATATTTATGCTGCCT
CUC2-ChIP #2	GATTTGCAACCTGTAACTTC	TGTCAGCACAGTACATGATT
CUC2-ChIP #3	TCTTCTCTACGACTTTCTGG	TAAGAAGAAAGATCTAAAGCTTTT G
ACT7-ChIP	CGTTTCGCTTTCCTTAGTGTT AGCT	AGCGAACGGATCTAGAGACTCAC CTTG