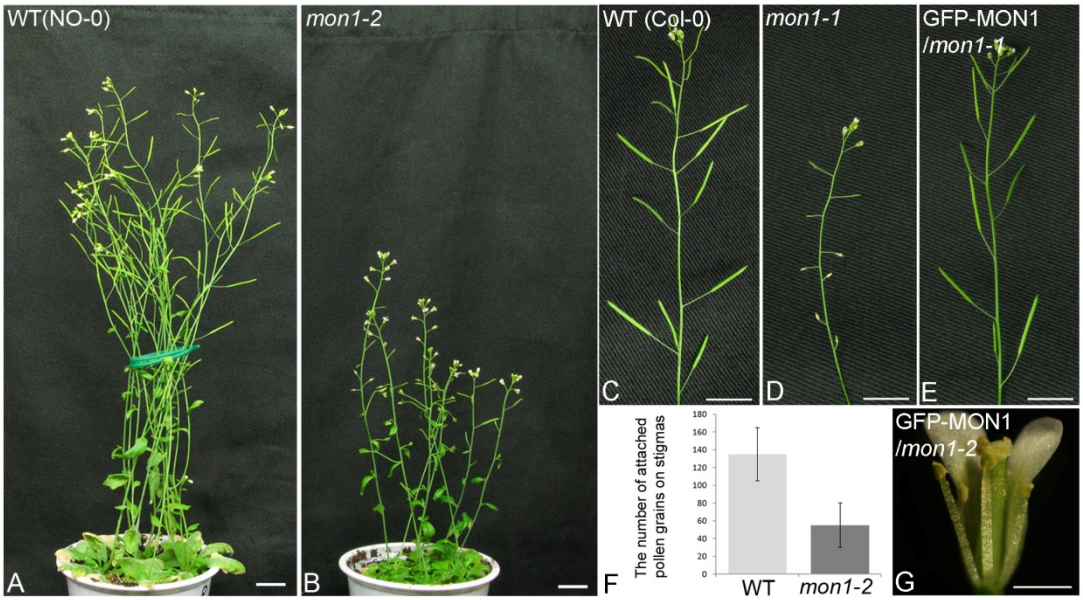
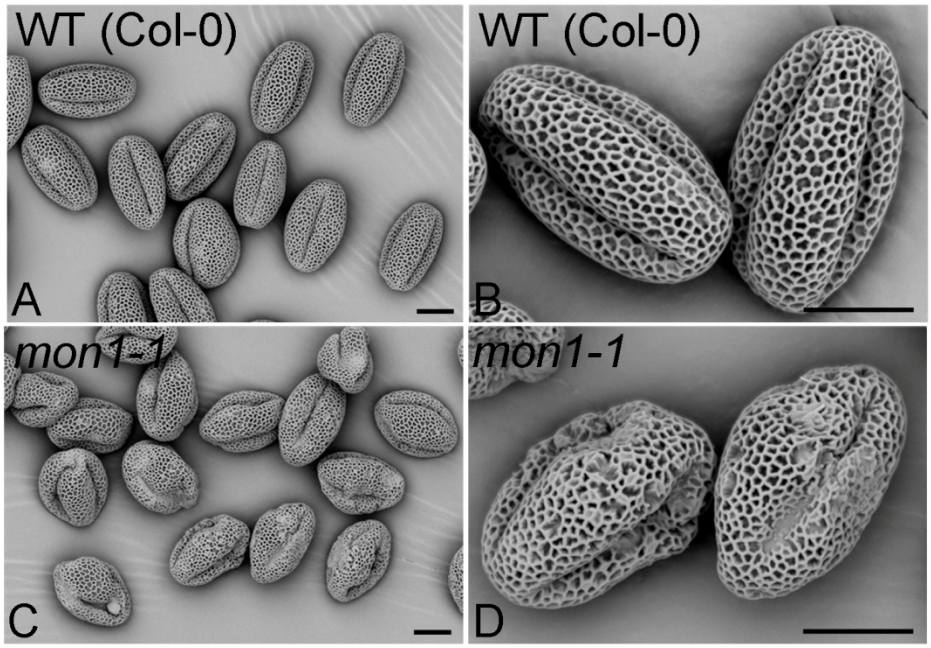


Supplemental Figure S1. MON1 is essential for normal plant growth and reproduction.

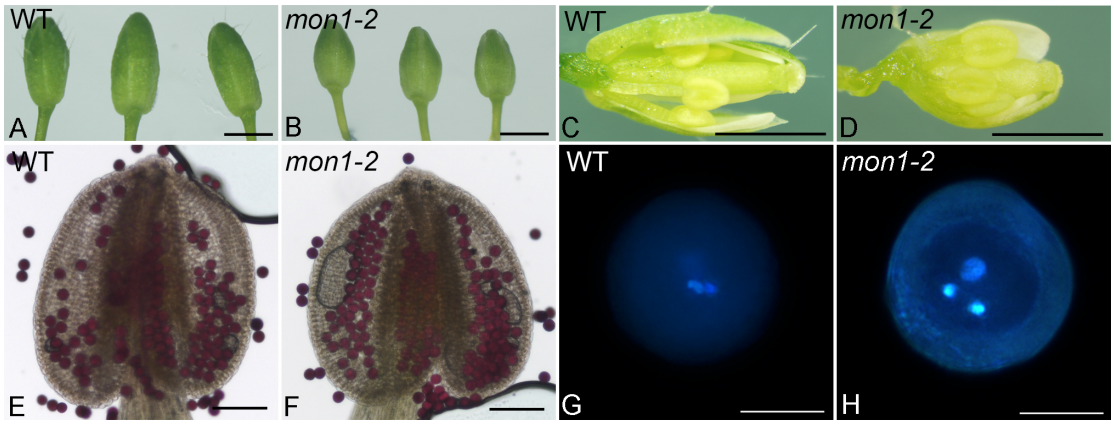
A to E. The *mon1* mutants were dwarf with reduced fertility and GFP-MON1 recovered this phenotype. Bars = 1 cm.

F and G. Compared to the wild-type, less pollen grains from *mon1-2* were attached to stigmas when hand-pollinated to wild-type stigma (F). GFP-MON1/*mon1-2* showed complete recovery (G). Bars = 1 mm in (G).



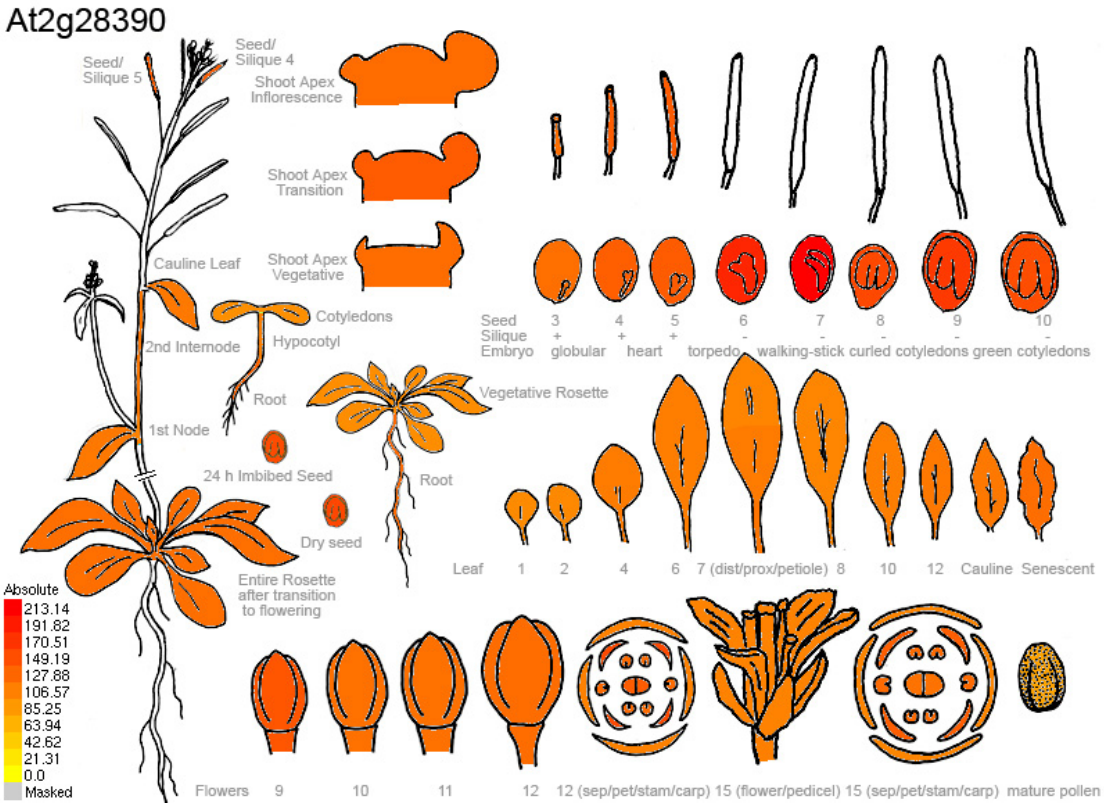
Supplemental Figure S2. *MON1* mutation causes defective pollen coat formation.

SEM images showed *mon1-1*$^{-/-}$ mutants (C and D) had abnormal pollen coats comparing to the wild-type (A and B). Bars = 10 μm.



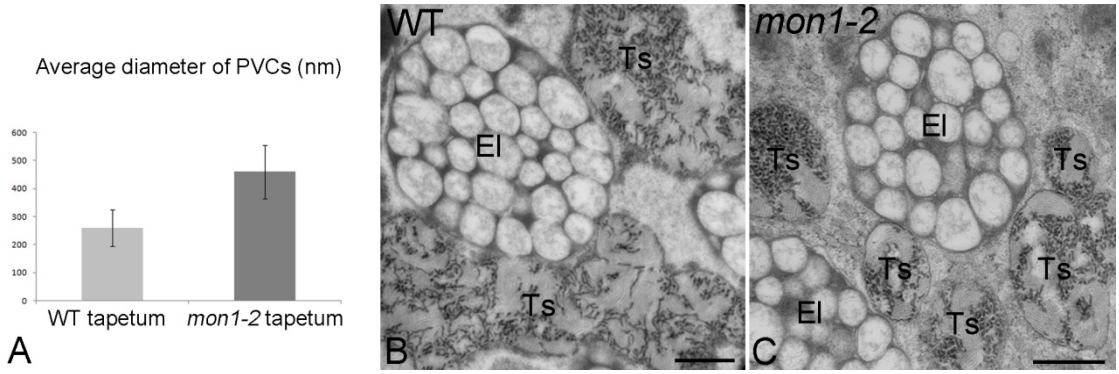
Supplemental Figure S3. The *mon1* mutants show normal floral organs and pollen viability.

Morphology of *mon1-2* flowers compared with wild-type (A-D). No difference of floral organs (A to D) and pollen viability (E to H) between wild-type and *mon1* mutants was observed based on Alexander and DAPI staining of mature pollen from wild-type and *mon1-2*. Bars = 1 mm in (A) to (D) and 10 μm in (E) to (H).



Supplemental Figure S4. *Arabidopsis* eFP browser report shows *MON1* gene expression in different plant organs and tissues.

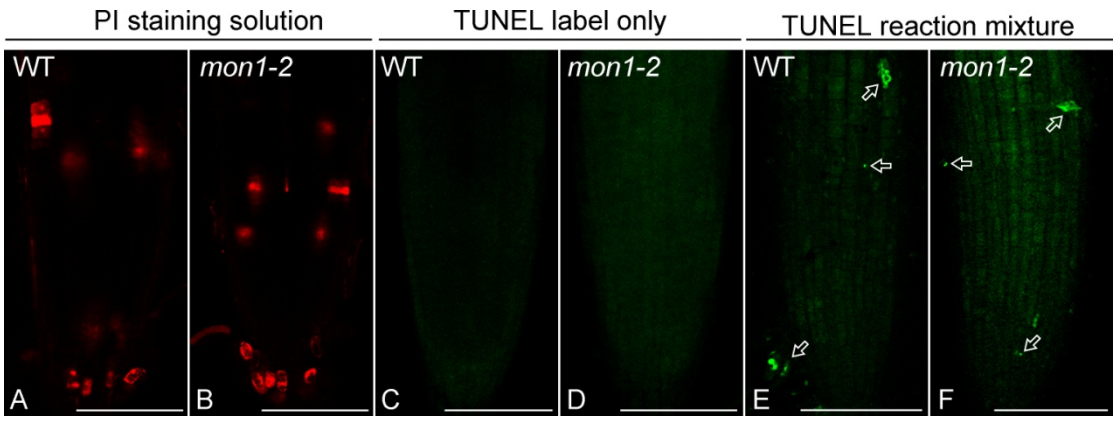
MON1 showed relative high expression in various organs and tissues, including the flower and pollen.



Supplemental Figure S5. The *mon1* tapetum shows normal tapetosomes and elaioplasts, but enlarged PVCs in TEM analysis.

A. Statistical analysis of the PVC size in wild-type and *mon1-2* tapetal cells. Each column represents the mean of at least 20 PVCs. Error bars indicate SD.

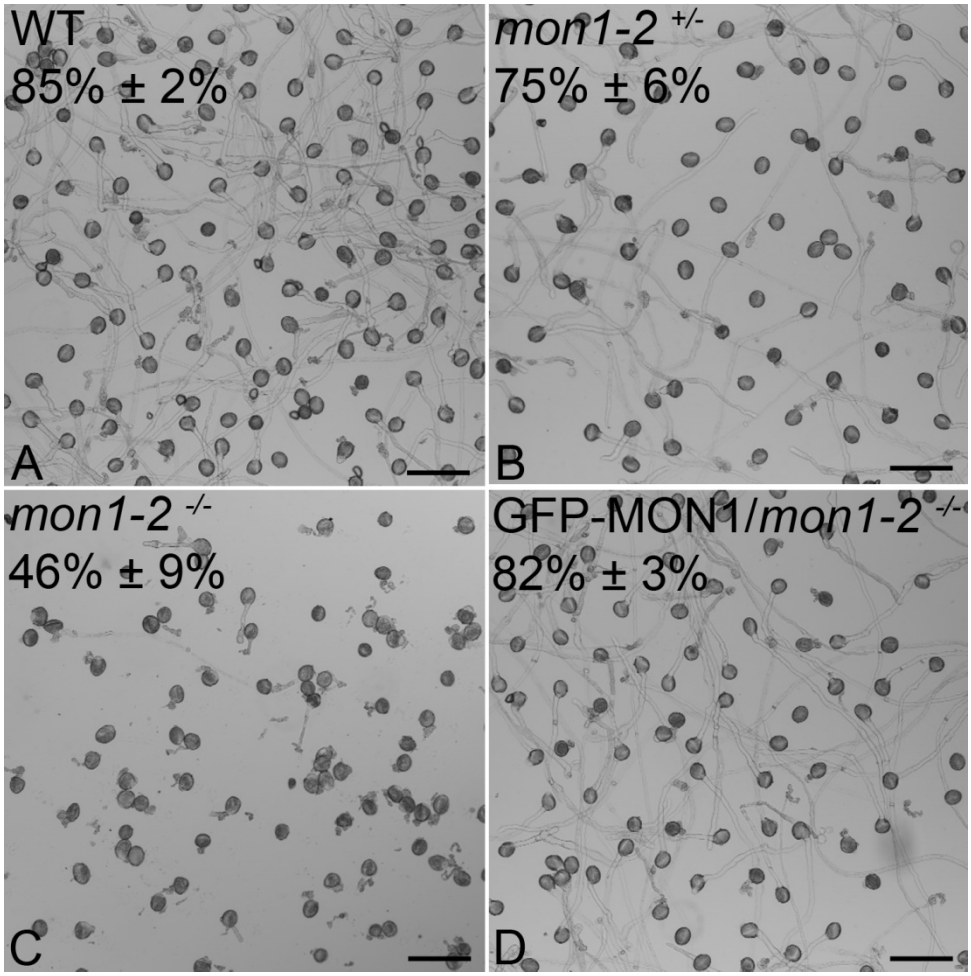
B and C. TEM images showed normal tapetosomes and elaioplasts in tapetal cells from wild-type and *mon1*. Ts, tapetosome; El, elaioplast. Bars = 500 nm.



Supplemental Figure S6. *MON1* mutation does not significantly alter the PCD in root cap.

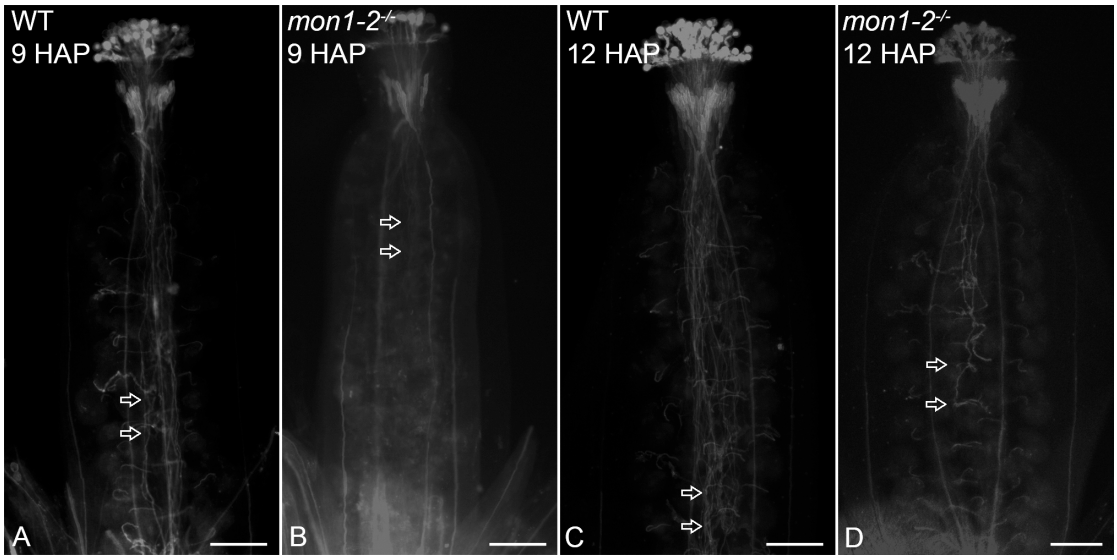
A and B. PI stained lateral root cap cells from WT and *mon1-2* were shown. Bars = 100 μm.

C to F. Wild-type and *mon1-2* roots were incubated with TUNEL label solution or TUNEL reaction mixture before imaging. The TUNEL positive lateral root cap cells that corresponded to two PCD zones in wild-type and *mon1-2* were indicated by the arrow. Bars = 100 μm.



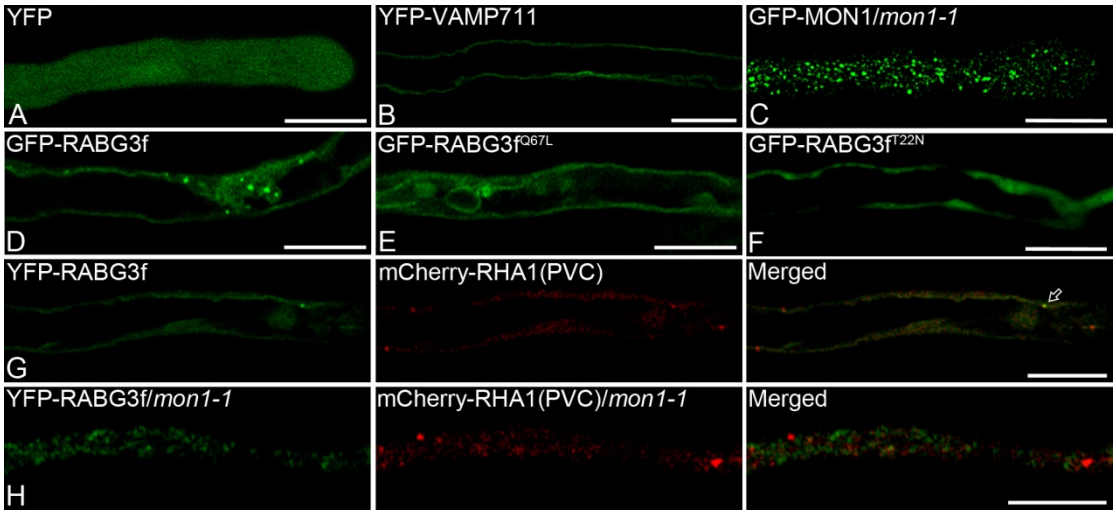
Supplemental Figure S7. *MON1* mutation causes defective pollen germination *in vitro*.

The representative images and statistical analysis indicated that homozygous *mon1* mutants (C) showed decreased germination rate comparing to the wild-type (A), heterozygous *mon1* mutants (B) and GFP-MON1/*mon1-2*$^{-/-}$ (D) *in vitro.* Bars = 100 μm.



Supplemental Figure S8. The *mon1-2* mutants show retarded pollen tube growth *in vivo*.

Aniline blue staining of emasculated wild-type pistils hand-pollinated with *mon1-2* pollen (B and D) showed retarded pollen tube growth of *mon1-2* at indicated times, comparing to the wild-type (A and C). The arrows indicated the pollen tube front. HAP, hours after pollination. Bars = 100 μm.



Supplemental Figure S9. Loss of function of *MON1* impairs the Rab5-to-Rab7 transition on PVCs in germinated pollen tubes, resulting in their retarded growth.

A to C. In germinated pollen tube, YFP and YFP-VAMP711 showed cytosol and tonoplast pattern respectively, while GFP-MON1 showed punctate pattern. Bars = 10 μm.

D to F. In germinated pollen tube, GFP-RABG3f localized to endosomes and tonoplast, GFP-RABG3f^{Q67L} localized to tonoplast and GFP-RABG3f^{T22N} showed cytosol pattern. Bars = 10 μm.

G. In germinated pollen tube, YFP-RABG3f localized to tonoplast and endosomes which were partially colocalized with mCherry-RHA1 positive PVCs (indicated by arrow). Bars = 10 μm.

H. In *mon1-1* mutant pollen tube, YFP-RABG3f localized to endosomes, which were separated from mCherry-RHA1 positive PVCs. Bars = 10 μm.

Supplemental Table S1. *MON1* mutation results in defective male transmission.

Experiment parent	F1 segregation		
Female × male	Genotype	Expected ratio	Observed
mon1-1$^{+/-}$ × WT	*MON1-1* $^{+/-}$: WT	1:1	49:53
WT × *mon1-1*$^{+/-}$	*MON1-1* $^{+/-}$: WT	1:1	42:62
mon1-2$^{+/-}$ × WT	*MON1-2* $^{+/-}$: WT	1:1	52:55
WT × *mon1-2*$^{+/-}$	*MON1-2* $^{+/-}$: WT	1:1	44:60
mon1-1$^{+/-}$ × *mon1-1*$^{+/-}$	R:S[a]	3:1	346:152
mon1-2$^{+/-}$ × *mon1-2*$^{+/-}$	R:S[b]	3:1	316:128

[a]R, Kanamycin resistant; S, Kanamycin sensitive.

[b]R, Hygromycin resistant; S, Hygromycin sensitive.

Supplemental Table S2. Primers used in this study.

Construct name	Primer name	Primer sequence (from 5' start to 3' end)
MON1	Forward	GAATTGATTTAGGTGACACTATAGCGAATTTAGCGGTGGCGGCGAT
	Reverse	GAATTGTAATACGACTCACTATAGGGGCTGCGAGGCAGTGTCACCATC
GFP-RABG3f	Forward	GGGTCTAGAATGCCGTCCCGTAGACGTACCCT
	Reverse	GGGCTCGAGTTAGCATTCACACCCTGTAGACCTCT
RD21-YFP	Forward	GGGTCTAGAATGGGGTTCCTTAAGCCAACCATGGCGA
	Reverse	GGGCTCGAGGGCAATGTTCTTTCTGCCTTGTGACCA
RD19-YFP	Forward	GGGTCTAGAATGGATCGTCTTAAGCTTTATTTCTCCGTTT
	Reverse	GGGCTCGAGATGGGCGGTGGTTGAGACGGTGGCT
RDL1-YFP	Forward	GGGGGATCCATGGCTCCTTCAACAAAAGTTCTCTCTTTACTT
	Reverse	GGGCTCGAGAACACTGCTGATAGTATTTCCACGAACCGGGTT
AT4G32940-YFP	Forward	GGGTCTAGAATGGCCACAACGATGACACGTGTCTC
	Reverse	GGGCTCGAGTGCACTGAATCCACGGTTAAGCGAGCTC
γVPE-YFP	Forward	GGGTCTAGAATGGCCACAACGATGACACGTGTCTCCGT
	Reverse	GGGCTCGAGTGCACTGAATCCACGGTTAAGCGAGCTC