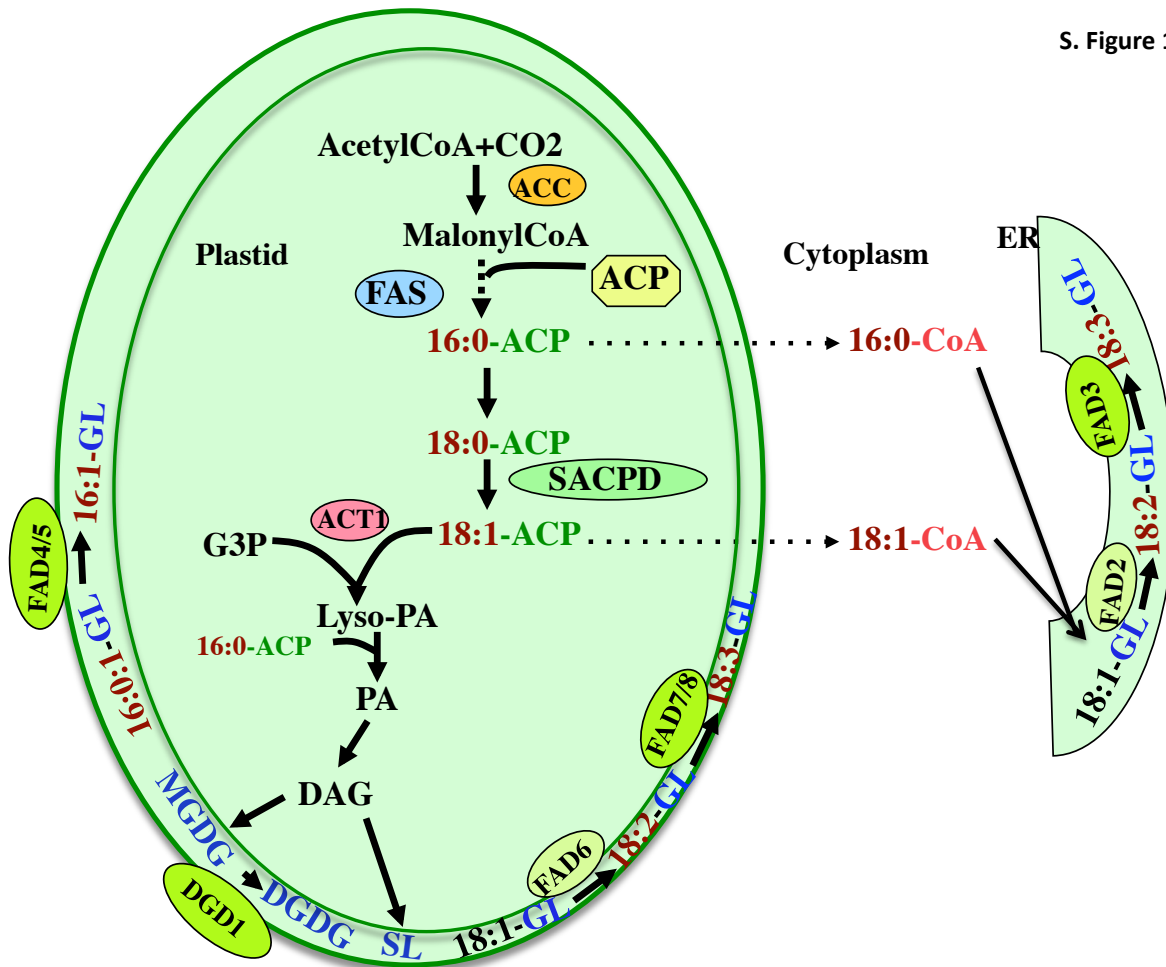
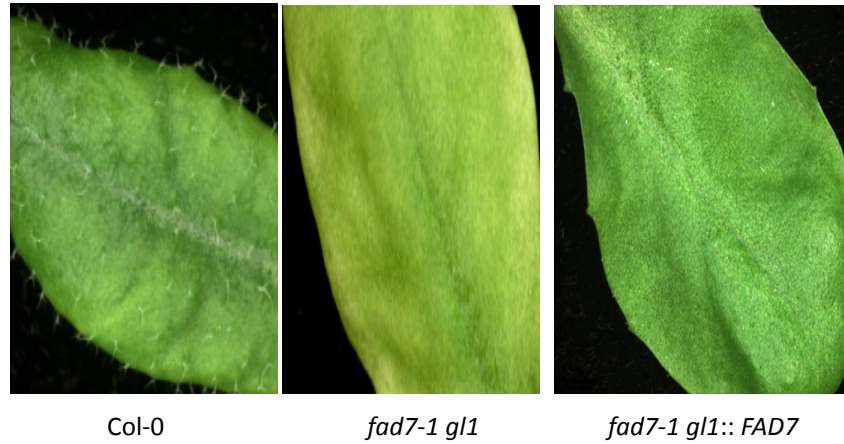


S. Figure 1

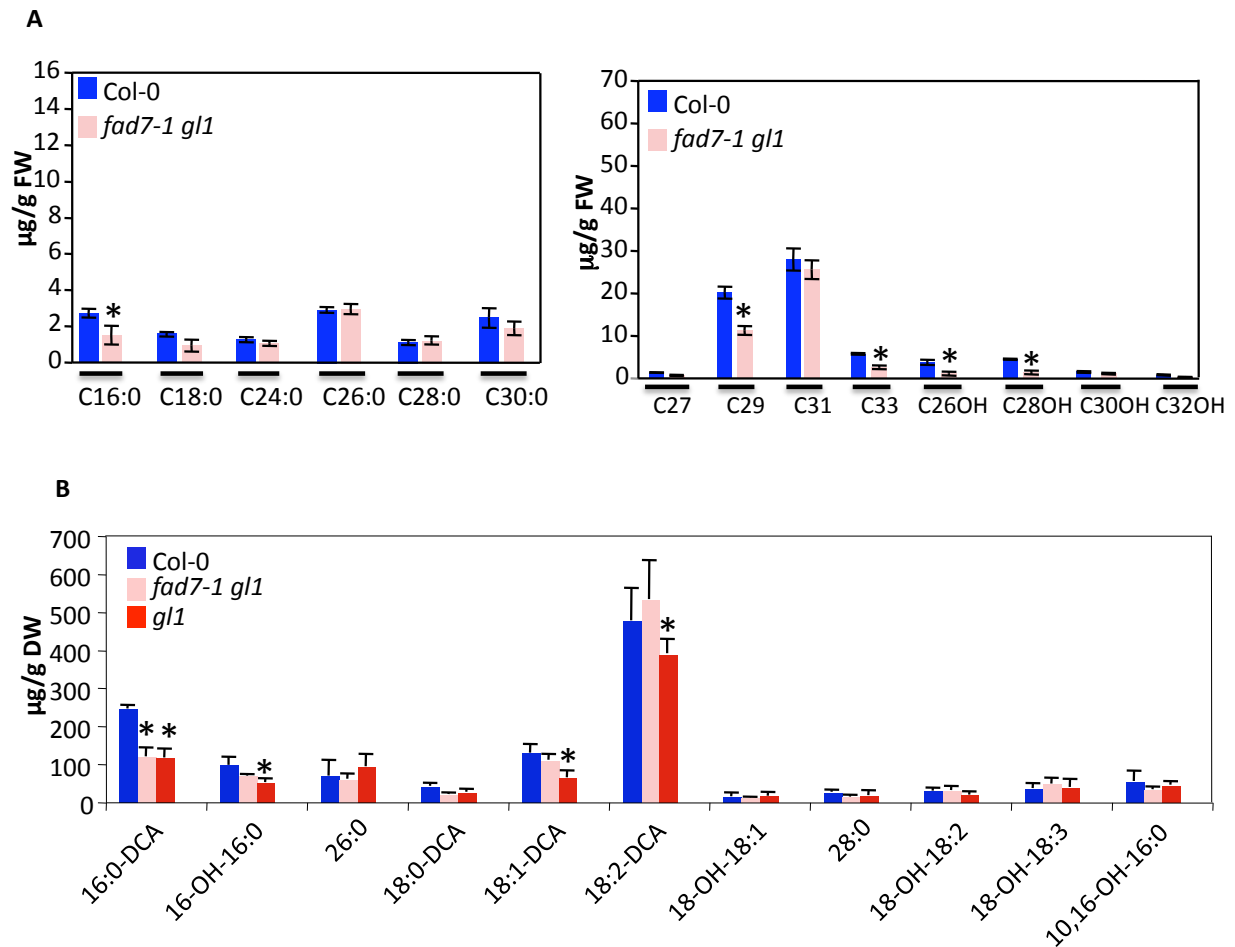


S. Figure 1. An Abbreviated scheme for fatty acid and lipid biosynthesis. *De novo* FA biosynthesis from acetyl-CoA occurs exclusively in the plastids of all cells (represented by oval). Acetyl-CoA carboxylase (ACC) and fatty acid synthase (FAS) complex are key enzymes involved in biosynthesis of palmitic acid (16:0). Upon elongation to stearic acid (18:0), this FA undergoes desaturation to oleic acid (18:1). Desaturation of stearic acid (18:0)-ACP to 18:1-ACP catalyzed by the *SSI2/FAB2* encoded stearyl-acyl carrier protein (ACP) desaturase (SACPD), is one of the key steps in the FA biosynthesis pathway that regulates levels of unsaturated FAs in the cell. The 18:1-ACP generated in this reaction enters the prokaryotic pathway through acylation of glycerol-3-phosphate (G3P) and this reaction is catalyzed by the *ACT1*-encoded G3P acyltransferase. The 18:1-ACP is also exported out of plastids as a CoA-thioester to enter the eukaryotic pathway. Desaturation of 18:1 present on membrane glycerolipids (GL) is catalyzed by *FAD2* and *FAD6* encoded ω 6 desaturases that are present on endoplasmic reticulum (ER) or plastid envelop, respectively. Desaturation of 18:2 present on membrane GL is catalyzed by *FAD3* and *FAD7/ FAD8* encoded desaturases that are present on ER and plastid respectively. Desaturation of 16:0 to 16:1 is catalyzed by plastidal localized desaturases *FAD4* and *FAD5*. Symbols for various components are: Lyso-PA, acyl-glycerol 3-phosphate; PA, phosphatidic acid; MGDG, monogalactosyldiacylglycerol; DGDG, digalactosyldiacylglycerol; SL, sulfolipid; DAG, diacylglycerol.

S. Figure 2

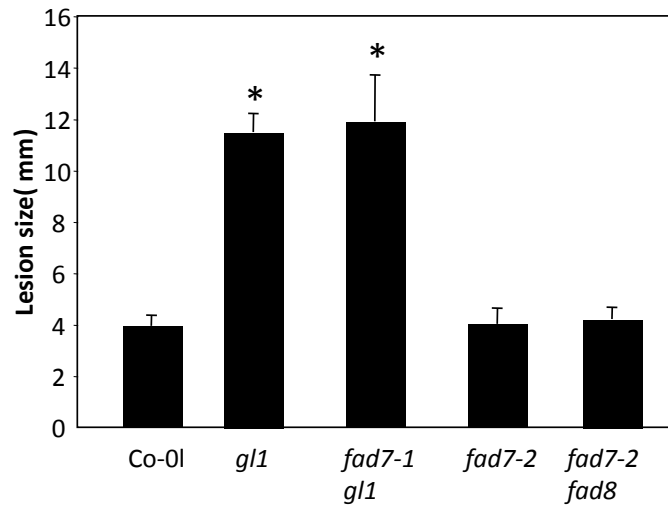


S. Figure 2. Leaves from indicated genotypes showing presence or absence of trichomes. Five different transgenic lines expressing genomic copy of *FAD7* in *fad7-1 gl1* background were tested and all showed absence of trichomes.



S. Figure 3. Cuticular wax and cutin monomer profiles. (A) Analysis of wax components from leaves of indicated genotypes. 16:0-24:0 are FAs, C27-C33 are alkanes, C26-OH-C32-OH are primary alcohols. (B) Analysis of lipid polyester monomer content of four-week-old plants. Error bars in A and B represent SD. Asterisks in A and B denote a significant difference with wild-type (Col-0) (*t* test, $P < 0.05$). Symbols for various components are: 16:0-DCA, 1,16-hexadecane dioic acid; 16-OH-16:0, 16-hydroxyhexadecanoic acid; 10,16-OH-16:0, 10,16-dihydroxyhexadecanoic acid; 18:0-DCA, 1,18-octadecane dioic acid; 18:1-DCA, 1,18-octadecene dioic acid; 18:2-DCA, 1,18-octadecadiene dioic acid; 18-OH-18:1, 18-hydroxyoctadecenoic acid; 18-OH-18:2, 18-hydroxyoctadecadienoic acid; 18-OH-18:3, 18-hydroxyoctadecatrienoic acid.

S. Fig. 4

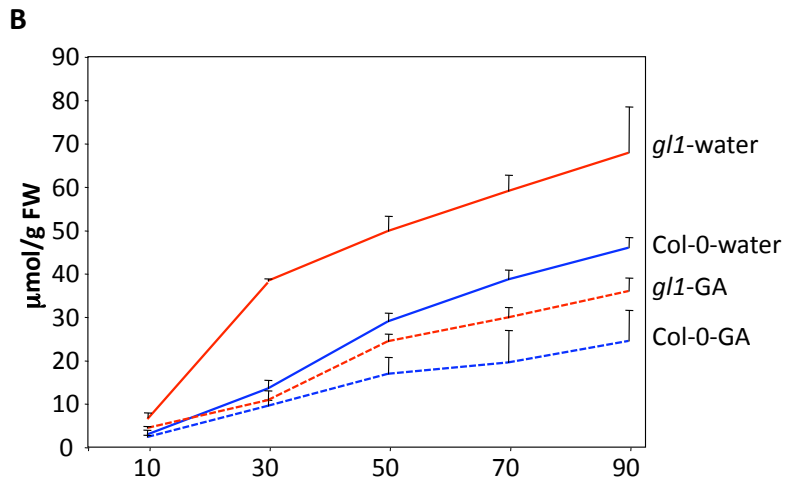


S. Figure 4. Response of *gl1* to necrotrophic pathogen *B. cinerea*. Lesion size in spot-inoculated genotypes. The plants were spot-inoculated with 10^6 spores ml^{-1} of *B. cinerea* and the lesion size was measured from 20-30 independent leaves at 6 dpi. Statistical significance was determined using Student's *t*-test. Asterisks indicate data statistically significant from that of control (Col-0) ($P < 0.05$). Error bars indicate SD.

S. Fig. 5

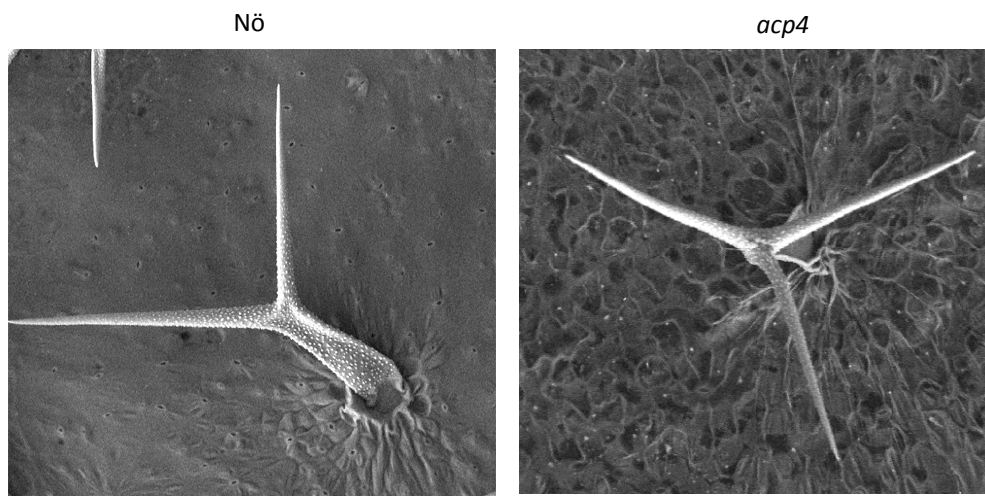


S. Figure 5. Leaves of indicated genotypes showing absence of trichomes on *gl3* and *ttg1* plants.



S. Figure 6. Effect of GA treatment on trichome formation, leaf size and chlorophyll leaching. (A) Leaves of Col-0 and *gll* plants treated with water or GA. GA treatment increased the leaf size but did not induce trichome formation on *gll* plants. scale bars, 2 cm. (B) A time-course measurement of chlorophyll leaching in Col-0 and *gll* plants treated with water or GA.

S. Fig. 7



Supplemental Figure 7. Scanning electron micrographs showing trichome on adaxial surface of leaves from Nö and *acp4* plants. Holes in *acp4* leaf indicate ruptured cuticle.