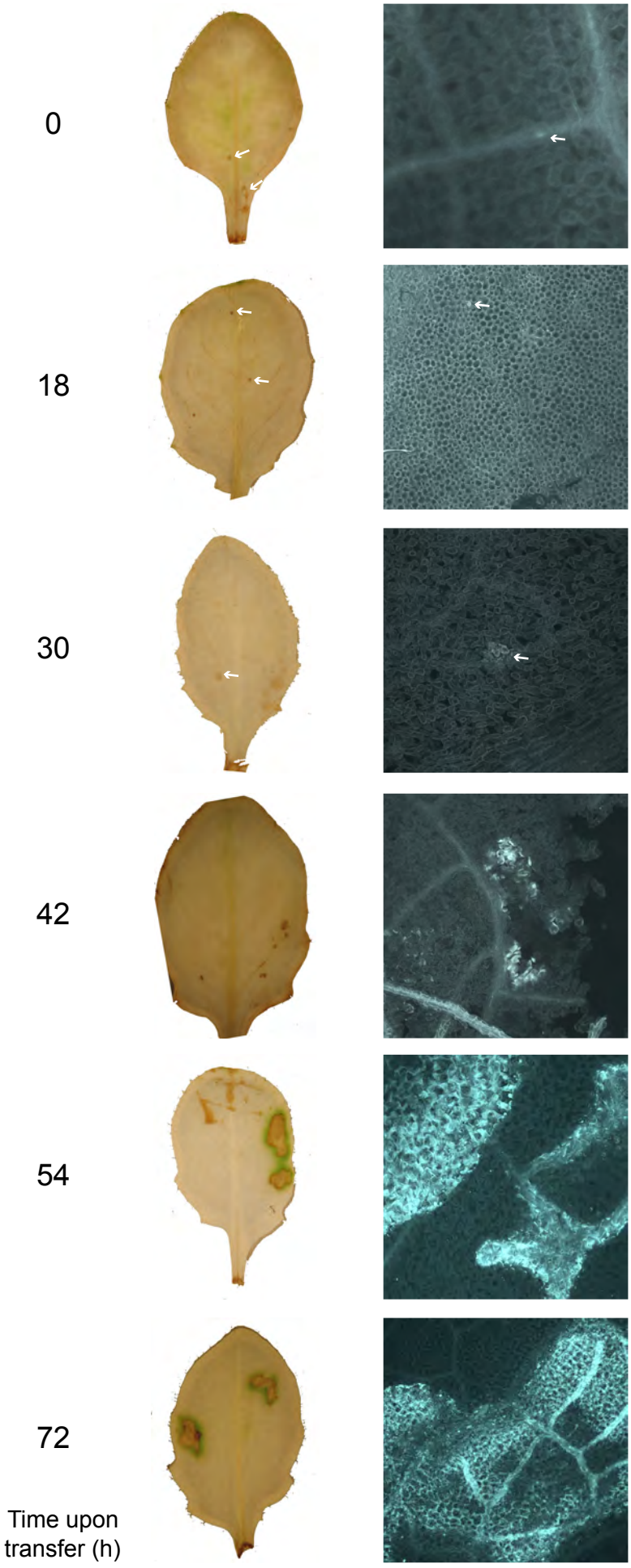

Figure SI1

Transfer of *aca4/11* to a non-suppresing growth medium induces an accumulation of ROS and the apparition of callose deposits. Plants grown hydroponically in an anion suppression condition (showing no visible lesions) were transfered at t = 0h into a standard solution. ROS levels (full leaves pictures) and callose deposition (leaves microscopy pictures- 1,3 mm wide) were tracked by DAB and aniline blue staining, respectively, during 54 to 72h. ROS can be visualized as dark brown dots, and callose deposits as brighter regions (see arrows).

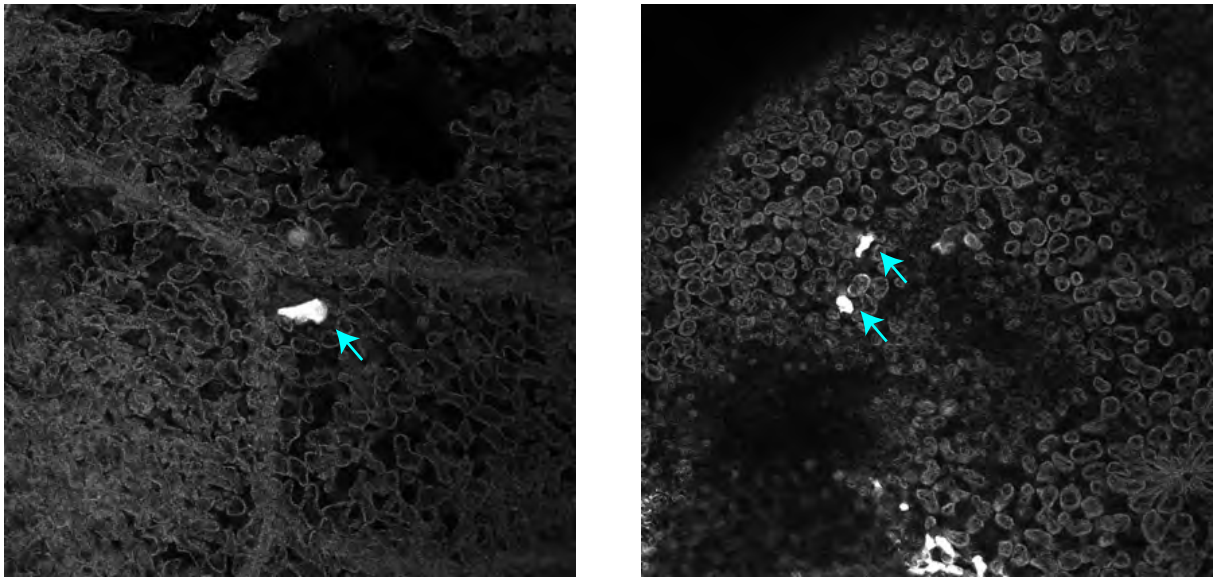

Figure SI2

Lesions initials in *aca4/11* occurs predominantly in mesophyll cells. Leaves of *aca4/11,* transfered from an anion suppression solution to a standard hydroponic solution, were stained with aniline blue for callose deposition. The figure presents two examples of optical sections of leaves (size: 1,3 mm) taken at 18h (left) or 30h (right) after transfer. Arrows indicate aniline blue-stained cells considered as micro-lesions. Globular shaped spaced cells correspond to mesophyll cells while closed round-shaped cells are considered as parenchyma.

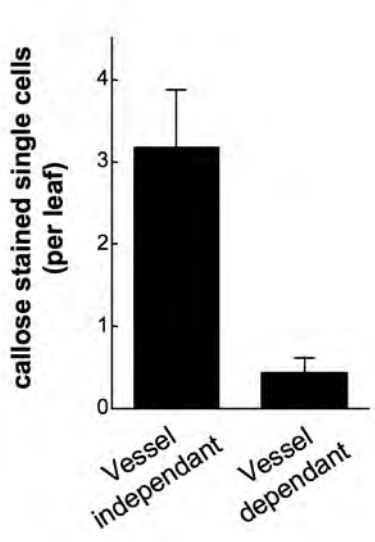

Figure SI3

Lesions and vessels localization were compared. Lesions initials stained with aniline blue (see Figure 4 and 5) appearing within 25mm of a vessel were considered as vessel associated. Average number of lesion initials per leaf and vessel dependent lesion initials for t=18 and 30h are presented (± SEM, n=16).

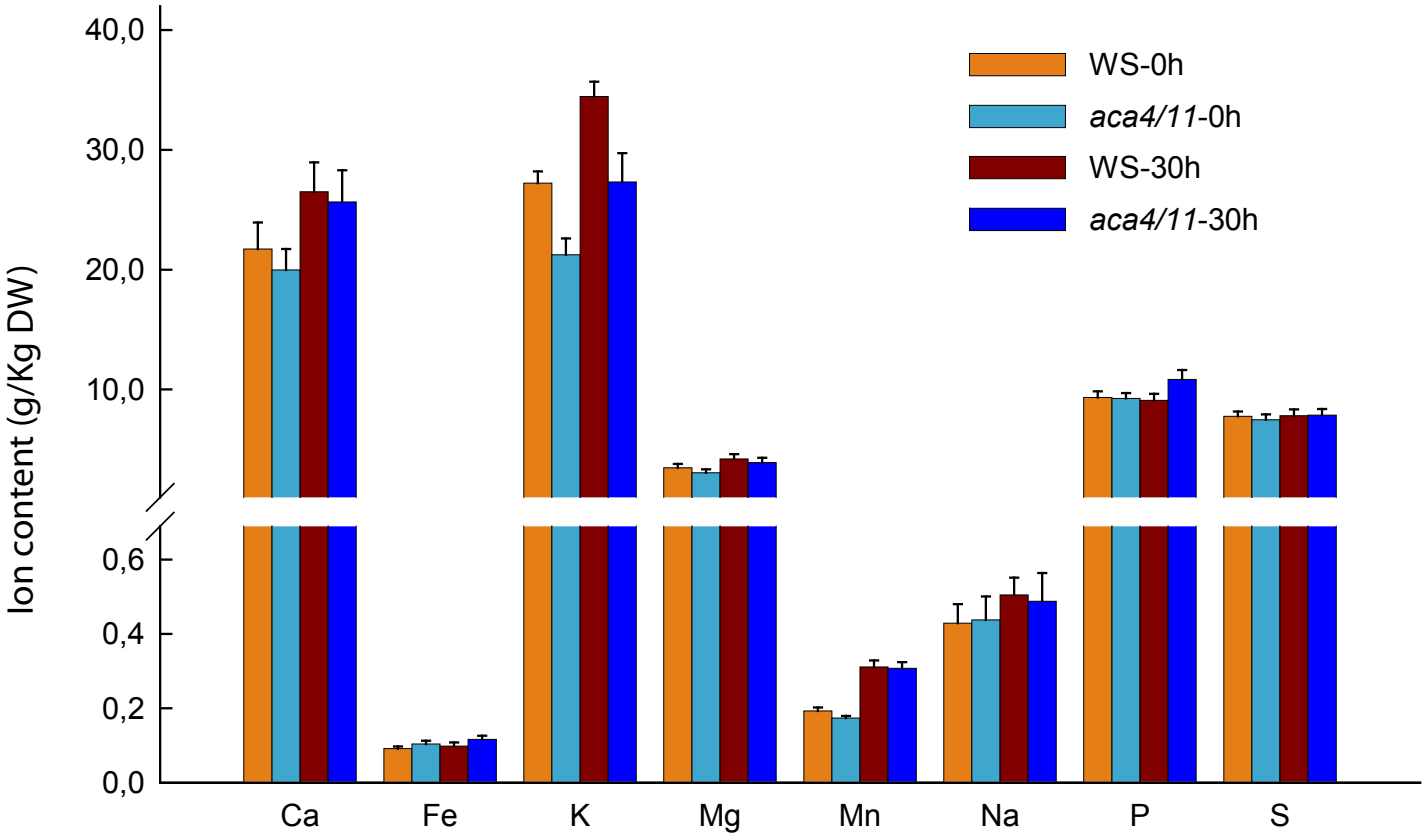

Figure SI4

Ion analysis measurements by ICP-AES providing the content of major ions in *aca4-1/11-1* and WS plants grown in standard hydroponic solution supplemented with 15mM NH_4NO_3 (lesion suppressed conditions), or in plants grow in parallel and transferred for 30h to a "lesion inducing solution" (standard hydroponic solution) (see Figure 4). No significant difference between WS and *aca4-1/11-1* were found, except a 20% reduction ($p<0.05$) for K in the double mutant in all conditions.

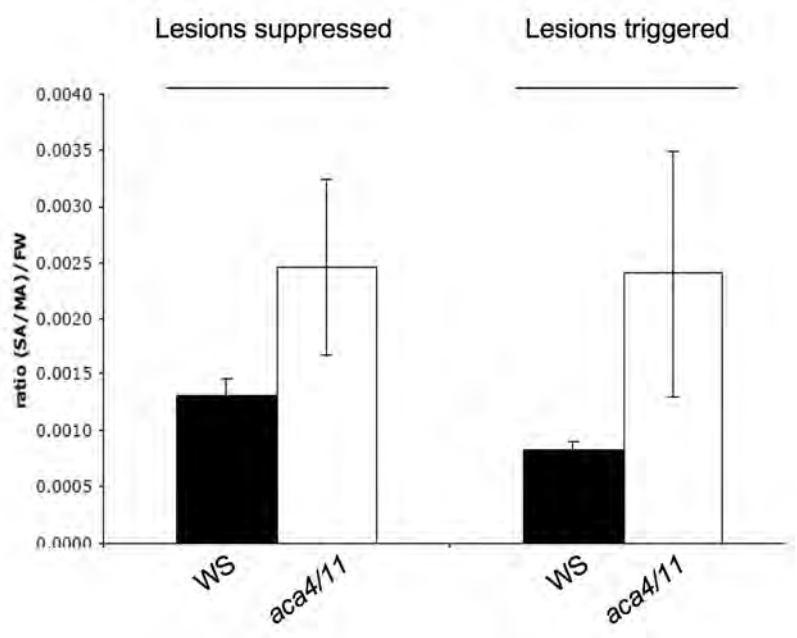

Figure SI5

Relative SA levels in *aca4/11* upon lesion induction.
The lesion induction was conducted similarly to fig. 4. Metabolites were extracted with chloroform and SA was measured by GC-MS after derivatization. SA levels are expressed relatively to the internal control mandelic acid (MA, added during the extraction) and rosette fresh weight (FW)

Figure SI6: DNA sequence of the *ACA11-GFP* contructs.

ps1657, *ACA11promoter:: ACA11-GFP* (The promoter is cloned between a unique Sbf1 site at 2623 and a unique Xho1 ste at 4758. The ACA11-GFP cDNA coding sequence is cloned between Xho1 at 4758 and a unique SacI at 8626.

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ps1658, *35s:: ACA11-GFP* (The 35s promoter is cloned between a Sbf1 site at 2642 and a unique Xho1 ste at 3533. The ACA11-GFP cDNA coding sequence is cloned between Xho1 at 3533 and a unique SacI at 7401.

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