Research Report

Short title: SA and JA modulate the ROS wave

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Jasmonic acid and salicylic acid modulate systemic reactive oxygen species signaling during stress responses

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One-sentence summary: An antagonistic interaction between salicylic acid and jasmonic acid attenuates reactive oxygen species accumulation in local and systemic tissues during plant responses to light stress or wounding.

ABSTRACT

Plants can send long-distance cell-to-cell signals from a single tissue subjected to stress to the entire plant. This ability is termed 'systemic signaling' and is essential for plant acclimation to stress and/or defense against pathogens. Several signaling mechanisms are associated with systemic signaling, including the reactive oxygen species (ROS) wave, calcium wave, hydraulic wave, and electric signals. The ROS wave coordinates multiple physiological, molecular, and metabolic responses among different parts of the plant and is essential for systemic acquired acclimation (SAA) to stress. In addition, it is linked with several plant hormones, including jasmonic acid (JA), salicylic acid (SA), and abscisic acid (ABA). However, how these plant hormones modulate the ROS wave and whether they are required for SAA is not clear. Here we report that SA and JA play antagonistic roles in modulating the ROS wave in Arabidopsis (*Arabidopsis thaliana*). While SA augments the ROS wave, JA suppresses it during responses to local wounding or high light (HL) stress treatments. We further show that ethylene and ABA are essential for regulation of the ROS wave during systemic responses to local wounding treatment. Interestingly, we found that the redox-response protein NONEXPRESSOR OF PATHOGENESIS RELATED PROTEIN 1 (NPR1) is required for systemic ROS accumulation in response to wounding or HL stress, as well as for SAA to HL stress. Taken together, our findings suggest that interplay between JA and SA might regulate systemic signaling and SAA during responses of plants to abiotic stress or wounding.

INTRODUCTION

Plants grow, develop, and reproduce in a dynamic environment that subjects them to rapid changes in conditions such as light intensity, humidity, temperature, and/or the presence of pathogens or pests (Kollist et al., 2019). To successfully thrive in their environment, plants need to rapidly respond and acclimate to these changes (Zandalinas et al., 2019; Zandalinas et al., 2020a). Although occasionally the entire plant is simultaneously exposed to the changing environmental conditions, in many instances only a single tissue of the plant (termed 'local' tissue) will sense the different stress conditions before the rest of the plant will. In such instances, the local tissue that first senses the change in environmental conditions will send a rapid systemic signal that spreads to the entire plant within minutes and activates defense and acclimation mechanisms in all other parts of the plant (termed 'systemic tissue'), often before the change in environmental conditions will reach them (Kollist et al., 2019; Zandalinas et al., 2020b). This process is known as 'rapid systemic signaling' and is essential for the systemic acclimation (termed 'systemic acquired acclimation'; SAA) of plants to different abiotic stresses such as excess light or heat stress (Suzuki et al., 2013; Zandalinas et al., 2019; Zandalinas et al., 2020a). In addition, it is essential for the systemic wound response (SWR) to mechanical wounding, that is in many instances associated with insect attack and herbivory (Toyota et al., 2018; Farmer et al., 2020). The rapid activation, or priming, of the entire plant, that occurs within minutes of the sensing of stress by a single tissue (driven by rapid systemic signaling), serves an important role in plant acclimation, triggering many of the slower acclimation responses that include the activation of multiple gene networks and adjustments of metabolism, that may take tens of minutes to hours (Kollist et al., 2019; Mittler et al., 2022).

Over the years, several different rapid systemic signals were identified in plants (Miller et al., 2009; Christmann et al., 2013; Toyota et al., 2018; Farmer et al., 2020; Fichman and Mittler, 2021a; Fichman and Mittler, 2021b). These include the reactive oxygen species (ROS) wave, the calcium wave, electric signals, the redox wave, and hydraulic waves. While electric, calcium, and hydraulic signals were found to be dependent on the function of the glutamate receptor-like (GLR) 3.3 and 3.6 channels (Toyota et al., 2018; Farmer et al., 2020; Fichman and Mittler, 2021a), the ROS wave was found to be dependent on the function of the respiratory burst oxidase homolog (RBOH) proteins (RBOHD and RBOHF), and the ROS sensor H_2O_2-induced

Ca^{2+} increases 1 (HPCA1), in Arabidopsis (*Arabidopsis thaliana*) (Miller et al., 2009; Zandalinas et al., 2020b; Fichman et al., 2022). In addition, many of the different rapid systemic signals were found to be mediated through the plant vascular system and to be dependent on each other (Farmer et al., 2020; Zandalinas et al., 2020b; Zandalinas and Mittler, 2021). However, differences between rapid systemic signaling in response to excess light stress (high light; HL) and mechanical injury were recently reported (Fichman and Mittler, 2021a), demonstrating that different local stimuli may trigger different combinations of these waves that are regulated by different sets of proteins.

In addition to coordinating systemic transcriptomic and metabolic responses of plants to stress, the ROS wave was recently found to coordinate the systemic stomatal responses of different leaves to a local exposure of HL, heat stress (HS), or injury (Devireddy et al., 2018; Devireddy et al., 2020a; Devireddy et al., 2020b; Zandalinas et al., 2020a). The closure of stomata on treated leaves in response to HL or wounding, or stomatal opening of the local leaf in response to a localized HS, were therefore propagated to the rest of the plant within minutes, in a process that required the function of the ROS wave (Devireddy et al., 2018; Devireddy et al., 2020a; Zandalinas et al., 2020a). These findings prompted studies of the role of different plant hormones in rapid systemic responses to abiotic stress.

The plant hormones abscisic acid (ABA), salicylic acid (SA), and jasmonic acid (JA) were found to accumulate in local and systemic leaves of plants in response to a local HL or heat stress treatments (Suzuki et al., 2013; Devireddy et al., 2018; Zandalinas et al., 2020a). ABA accumulation was further found to be required for local and systemic SA and H_2O_2 accumulation, as well as for systemic stomatal aperture changes, in response to a local HL stress (Devireddy et al., 2018; Devireddy et al., 2020a). JA was found to be required for local and systemic stomatal aperture changes in response to a local HL stress, and SA was found to be required for systemic, but not local stomatal aperture changes, in response to a local HL stress (Devireddy et al., 2020a; Zandalinas et al., 2020a). Although the studies described above revealed a role for ABA, JA, and SA in rapid systemic signaling, many questions regarding their function remained unanswered. For example, it is unknown what is the role of ABA, JA, and SA in mediating SAA to HL stress. In addition, the role of ABA, JA and SA in systemic wound responses is unclear. Furthermore, the role of several other stress-related hormones, such as

ethylene (ET) and strigolactones (SLs) in rapid systemic responses is unknown. To address these questions, we studied the local and systemic accumulation of ROS in different mutants deficient in ABA, JA, SA, ET, and SL biosynthesis or signaling to HL stress or wounding, as well as the SAA response of these mutants to HL stress.

Here we report that JA and SA play antagonistic roles in regulating the ROS wave. While JA suppresses the ROS wave during responses to HL stress, or wounding, SA augments the intensity of the ROS waves in response to these treatments. We further show that ET plays a role in the regulation of the ROS wave in response to wounding (but not HL stress), and that SLs do not play a role in responses to these two stresses. Interestingly, we found that the redox-response protein NONEXPRESSOR OF PATHOGENESIS RELATED PROTEIN 1 (NPR1) is required for systemic (but not local) ROS accumulation in response to HL stress or wounding, as well as for local and systemic acclimation to HL stress. This finding suggests that redox changes mediated by the ROS wave (Fichman and Mittler, 2021b) could regulate transcript expression in systemic tissues through SA- or ROS-mediated redox regulation, and that JA antagonizes these responses. Taken together, our findings suggest that SA and JA play antagonistic roles in regulating the ROS wave and that this regulation may be partially mediated by NPR1 in systemic tissues.

RESULTS

ABA biosynthesis is required for systemic ROS responses to wounding and SAA to HL stress

We previously reported that ABA levels transiently accumulate in local and systemic leaves of plants subjected to a local treatment of HL or HS (Suzuki et al., 2013; Devireddy et al., 2018; Zandalinas et al., 2020a). We further revealed that ABA biosynthetic mutants are unable to trigger H_2O_2 accumulation and stomatal closure in local and systemic leaves in response to a local HL stress treatment (Devireddy et al., 2018; Devireddy et al., 2020a). However, whether ABA is required for systemic plant acclimation to HL is unknown. In addition, the measurements of H_2O_2 in local and systemic tissues in response to HL stress were not performed with our highly sensitive whole-plant live ROS imaging method (Fichman et al., 2019; Zandalinas and

Mittler, 2021; Fichman et al., 2022), and the role of ABA in local and systemic ROS responses to wounding is unknown. To address these questions, we subjected two different alleles of the ABA biosynthesis mutant *aba2* (*aba2-11* and *aba2-4*; mutations in AT1G52340 encoding XANTHOXIN DEHYDROGENASE) to a local treatment of HL stress or wounding and imaged ROS accumulation in live plants grown in soil. *aba2* mutants were previously reported to retain 10-20% of wild type ABA levels (González-Guzmán et al., 2002). As shown in Figures 1A and 1B, ABA biosynthesis was essential for systemic ROS accumulation of plants in response to a local treatment of HL stress. ABA biosynthesis was also essential for local and systemic ROS accumulation in response to a local wounding treatment. The finding that the *aba2-11* mutant accumulated low levels of ROS in local leaves in response to HL stress (Figure 1A) could suggest that residual ABA levels found in some of the *aba2* mutants (González-Guzmán et al., 2002) could be sufficient to drive the HL-mediated ABA-dependent ROS accumulation response in local leaves, but that this response is not sufficient to trigger systemic ROS accumulation. As shown in Figure 1C, D, the two *aba2* mutants were unable to acclimate their local and systemic leaves to HL stress, suggesting that ABA is required for plant acclimation to HL stress even if ROS levels are enhanced. The results presented in Figure 1 suggest that ABA plays a key role in local and systemic ROS responses to wounding, in systemic ROS accumulation in response to a local HL stress, and in local and systemic acclimation of plants to HL stress.

JA is involved in local and systemic ROS responses to HL stress and wounding, as well as in SAA to HL stress

We previously reported that JA accumulates in local and systemic leaves of plants subjected to a local treatment of HL stress, and that JA signaling is required for local and systemic stomatal responses of plants to wounding, HL, and HS (Devireddy et al., 2018; Devireddy et al., 2020a; Zandalinas et al., 2020a). However, the role of JA in plant acclimation to HL stress or systemic ROS responses to HL or wounding is unknown. To study the role of JA in rapid systemic signaling to HL stress and wounding we used two different mutants: The JA signaling mutant *coi1* that is deficient in JA perception (mutation in AT2G39940 encoding a protein containing Leu-rich repeats and a degenerate F-box motif), and the JA biosynthesis mutant *aos1* required for JA biosynthesis (mutation in AT5G42650 encoding ALLENE OXIDE SYNTHASE). *coi1*

was previously reported to contain high basal levels of JA (Stotz et al., 2011), and *aos1* was reported to contain almost no detectable levels of JA (Park et al., 2002). As shown in Figure 2A, the *coi1* mutant was deficient in local and systemic ROS accumulation in response to a local treatment of HL stress or wounding. In contrast, the *aos1* mutant was not (Figure 2B). As shown in Figure 2C, the *coi1* mutant was also deficient in local and systemic acclimation to HL stress. In contrast, the *aos1* mutant was not (Figure 2D). The results presented in Figure 2 suggest that JA sensing by COI1 could play a key role in local and systemic ROS responses to wounding and HL stress, as well as in SAA to HL stress. Alternatively, the high basal levels of JA in the *coi1* mutant may suppress the ROS wave and SAA via a COI1-independent mechanism (*e.g.,* by antagonizing SA). In contrast to our results obtained with *coi1*, suppressing JA levels in the *aos1* mutant appeared to only decrease ROS signal accumulation in response to HL stress and wounding, but had no effect on SAA to HL.

SA and NPR1 are required for local and systemic acclimation to HL stress, and NPR1 is required for systemic ROS accumulation in response to HL stress or wounding

We previously reported that SA accumulates in local and systemic leaves of plants subjected to a local treatment of HL stress, that SA is required for SAA to HS, and that SA is required for systemic stomatal responses to a local treatment of HL stress (Suzuki et al., 2013; Devireddy et al., 2018; Devireddy et al., 2020a; Zandalinas et al., 2020a). However, the role of SA in plant acclimation to HL stress or systemic ROS responses to HL or wounding is unknown. To study the role of SA in rapid systemic signaling to HL and wounding, we used the *sid2* mutant (mutation in AT1G74710 encoding ISOCHORISMATE SYNTHASE 1) that is deficient in SA accumulation (retains 5-10% of wild type SA levels; Nawrath and Métraux, 1999), and the *npr1* mutant that is deficient in SA sensing (Spoel et al., 2003; Tada et al., 2008; Zhou et al., 2015; Caarls et al., 2015; Withers and Dong, 2016; Chen et al., 2021). As shown in Figure 3A, local and systemic ROS accumulation were suppressed in the *sid2* mutant in response to HL stress. In contrast, only systemic ROS accumulation was suppressed in the *sid2* mutant in response to wounding (Figure 3A). As shown in Figure 3B, while local accumulation of ROS was not suppressed in the *npr1* mutant in response to a local HL stress or wounding, systemic ROS accumulation was suppressed in the *npr1* mutant in response to both treatments. As shown in

Figure 3C and 3D, local and systemic acclimation to HL stress were abolished in both the *sid2* and *npr1* mutants. The findings that NPR1 is required for systemic (but not local) ROS accumulation in response a local wounding or HL stress treatments (Figure 3B), as well as for local and systemic acclimation to HL stress (Figure 3D), suggest that the ROS wave is triggered in local tissues of the *npr1* mutant in response to local wounding or HL stress, but that it does not spread to systemic leaves and does not induce SAA to HL stress in local or systemic tissues. To test whether PATHOGENESIS RELATED 1 (PR1) expression is corresponding with this phenotype of the *npr1* mutant, we tested the expression of PR-1 in local and systemic leaves of WT and *npr1* plants subjected to a local HL stress or wounding treatment. As shown in Figure 3E, the expression of PR-1, that is associated with responses to pathogens, HL stress and ROS/redox (Spoel et al., 2003; Tada et al., 2008; Zhou et al., 2015), was suppressed in local and systemic leaves of the *npr1* mutant in response to a local treatment of HL stress or wounding. This finding demonstrates that NPR1 is required for systemic expression of PR-1 in response to wounding or HL stress. Taken together, the findings presented in Figure 3 suggest that SA is essential for local and systemic acclimation to HL stress, and that NPR1 could play a key role in ROS accumulation and signaling in systemic tissues of plants subjected to HL stress or wounding.

JA suppresses the ROS wave, while SA augments it in response to a local treatment of HL stress or wounding

JA and SA were previously found to play antagonistic roles in mediating the response of plants to pathogens and other stresses, and this role was partially linked to the activation and nuclear localization of NPR1 (*e.g.,* Spoel et al., 2003; Tada et al., 2008; Zhou et al., 2015; Caarls et al., 2015; Withers and Dong, 2016; Chen et al., 2021). Because our findings suggest that JA and SA could play antagonistic roles in mediating the ROS wave during responses to HL stress and wounding (Figures 2 and 3), we tested whether application of JA or SA would affect the ROS wave triggered by these stresses. For this purpose, we applied 20 μM JA or 1 mM SA to wild type plants via fumigation prior to local stress application and measured local and systemic ROS accumulation in the presence or absence of JA or SA. As shown in Figure 4A, application of JA prior to the local HL stress or wounding treatments suppressed the ROS wave response of plants

to these treatments. In contrast, as shown in Figure 4B, application of SA prior to the local HL stress or wounding treatments augmented the systemic ROS wave response of plants to these treatments. The findings presented in Figure 4 support our findings presented in Figures 2 and 3 and suggest that JA and SA play antagonistic roles in regulating the ROS wave response of plants; JA suppresses it, while SA promotes it.

Because the basal levels of JA and SA could play a key role in triggering the ROS wave, we measured the levels of JA, SA and ABA in the local and systemic leaves of untreated wild type and the *coi1*, *aos1-1*, *sid2* and *npr1-1* mutants. As shown in Supplementary Figure 1A, and in agreement with Stotz et al., (2011), the levels of JA were higher in untreated *coi1* plants, compared to untreated wild type plants. In contrast, the levels of JA were lower than that of control in local leaves of untreated *aos1-1* and *npr1-1* plants. As shown in Supplementary Figure 1B, the levels of SA were suppressed in the local or systemic leaves of the *coi1* and *sid2* mutants, further supporting the finding that *coi1* has higher levels of JA. Interestingly, as shown in Supplementary Figure 1C, the levels of ABA were higher than control in the local leaves of all untreated mutants.

Because JA was able to suppress the ROS wave in wild type plants subjected to a local treatment of HL stress or wounding (Figure 4A), and the ROS wave was not suppressed in the *aos1-1* mutant (Figure 2B), that has suppressed levels of JA (Supplementary Figure 1A), we tested whether application of JA to the *aos1-1* mutant will suppress the ROS wave in this mutant in response to a local treatment of HL or wounding. As shown in Figure 4C and 4D, prior treatment of the *aos1-1* mutant with JA suppressed the ROS wave triggered in this mutant in response to HL stress or wounding. This finding supported the role of JA in suppressing the ROS wave during responses to HL stress or wounding.

ET is essential for systemic ROS accumulation in response to local wounding, while SLs appear to not play a role in systemic responses to HL or wounding stresses

ET is involved in many stress responses of plants, as well as in different developmental processes (Gamble et al., 1998; Alonso et al., 1999). However, little is known about the role of ET in systemic ROS responses of plants to stress. To study the potential roles of ET in local and

systemic responses to HL stress and wounding we subjected two ET sensing mutants, *ein2* (mutation in AT5G03280) and *etr1* (mutation in AT1G66340), both transmembrane proteins required for triggering ET responses in Arabidopsis (Gamble et al., 1998; Alonso et al., 1999), to a local treatment of HL stress or wounding and measured local and systemic ROS accumulation. As shown in Figure 5A and 5B, ET sensing by EIN2 and ETR1 was not required for local or systemic ROS accumulation in response to HL stress. In contrast, ET sensing by these two proteins was required for systemic, but not local, ROS accumulation in response to wounding (Figure 5A, 5B). As shown in Figure 5C and 5D, both *etr1* and *ein2* could acclimate to HL stress. The findings presented in Figure 5A-D reveal that ET could be playing a role in systemic ROS accumulation in response to wounding.

Strigolactones play important roles in plant-pathogen interactions and developmental responses to abiotic stress (Saeed et al., 2017). To test whether SLs play an important role in local and systemic responses to HL stress and wounding, we used the SL mutants *max2* involved in SL sensing (mutation in AT2G42620 encoding an F-box leucine-rich repeat protein), and *max3* involved in SL biosynthesis (mutation in AT2G44990 encoding CAROTENOID CLEAVAGE DIOXYGENASE 7). As shown in Supplementary Figure 2, *max2* and *max3* were not deficient in their local or systemic ROS accumulation in response to wounding or HL stress, or in SAA to HL. These results suggest that SLs might not be involved in rapid responses to wounding or HL stress.

DISCUSSION

We previously reported that ABA, JA, and SA play important roles in regulating local and systemic stomatal responses to a local application of HL stress (Devireddy et al., 2018; Devireddy et al., 2020a; Zandalinas et al., 2020a). ABA, SA, and JA rapidly accumulate in local and systemic leaves of plants in response to a local HL or heat stress treatments (Suzuki et al., 2013; Devireddy et al., 2018; Zandalinas et al., 2020a), while JA rapidly accumulates in local and systemic leaves in response to wounding (Glauser et al., 2009). ABA is required for local and systemic SA and H_2O_2 accumulation, as well as for systemic stomatal aperture changes, in response to a local HL stress (Devireddy et al., 2018; Devireddy et al., 2020a), while JA is required for local and systemic stomatal aperture changes in response to a local HL stress, and

SA is required for systemic, but not local stomatal aperture changes, in response to a local HL stress (Devireddy et al., 2020a; Zandalinas et al., 2020a). The rapid accumulation of these hormones in local and systemic tissues in response to stress is thought to result from rapid synthesis (JA; Glauser et al., 2009), or release from conjugated/stored forms (SA/ABA; Suzuki et al., 2013; Kollist et al., 2019). Nevertheless, the role some of these hormones (*e.g.,* ABA) play in SAA to HL stress, or SWR is not clear. Our current study reveals an important function for ABA and SA in plant acclimation to HL stress, as well as in regulating the ROS wave in response to HL stress and wounding (Figures 1 and 3). In addition, we reveal a role for ABA and ET in systemic ROS responses to wounding (Figures 1 and 5). Interestingly, while ABA was required for local and systemic ROS responses to HL and wounding (that may involve ET responses; Cheng et al., 2009; please see below), ET signaling was only required for systemic ROS responses to wounding (Figures 1 and 5). This finding is in agreement with our previous findings that the systemic ROS response of plants to wounding is different than that to HL stress (*i.e.,* depended on different regulators and could occur through different plants tissues; *e.g.,* systemic ROS responses to HL are mediated trough the vascular system and may not require glutamate-like receptors 3.3 and 3.6, while systemic ROS responses to wounding are mediated through the vascular system or mesophyll cells and are dependent on glutamate-like receptors 3.3 and 3.6; Zandalinas et al., 2020b; Fichman and Mittler, 2021a; Zandalinas and Mittler, 2021). It is also possible that HL stress or wounding trigger different sources of ROS production (Fichman et al., 2021; Xiong et al., 2021), and that although both require ABA, local ROS responses to wounding do not require ET. Previous studies have shown that ABA and ET have antagonistic interactions and that in the *aba2* mutant some ET responses are suppressed (Cheng et al., 2009). This finding could explain why in the *aba2* mutant systemic ROS wave responses to wounding, that require ET signaling (Figure 5), are suppressed (Figure 1). Further studies are required to address the sources of local ROS produced during HL stress or wounding and their interactions with different plant hormones and other regulators (*e.g.,* phytochrome B; Fichman et al., 2021; Xiong et al., 2021). In addition, the role of the chloroplast, which is the initial site of ABA, SA, and other plant hormone biosynthesis during these responses, as well as the different plant tissues involved, need to be defined in future studies in different local and systemic tissues during responses to different stresses.

316 In contrast to SA and ABA, the involvement of JA in regulating the rapid systemic response of
317 plants to HL stress or wounding appears to be more complicated (Figures 2 and 4). JA was
318 initially shown to be required for local and systemic stomatal responses to HL stress (Devireddy
319 et al., 2018; Devireddy et al., 2020a), and as shown in Figure 2, JA sensing by COI1 is also
320 required for local and systemic ROS production and plant acclimation to a local treatment of HL
321 stress. However, because the *aos1* mutant that does not accumulate JA (Park et al., 2002;
322 Supplemental Figure 2) can still accumulate local and systemic ROS and acclimate to a local HL
323 stress treatment (Figure 2), it is possible that the role of COI1 in these responses is independent
324 of JA signaling. In this respect it should be noted that COI1 was found to have JA-independent
325 functions (*e.g.,* Stotz et al., 2011). An alternative possibility, that appears more plausible, is that
326 in the *coi1* mutant the basal levels of JA are high (due to a positive feedback loop on JA
327 synthesis; similar to what happens in the *abi1* mutant with ABA; Devireddy et al., 2018), and
328 that these high levels of JA antagonize SA function and cause the *coi1* mutant to not acclimate or
329 accumulate ROS. In this respect it should be noted that the *coi1* mutant was found to have high
330 basal levels of JA, supporting this possibility (Stotz et al., 2011; Supplementary Figure 2).
331 Moreover, treatment of the *aos1-1* mutant with JA, suppressed the ROS wave in this mutant in
332 response to HL or wounding (Figures 4C and 4D), further suggesting that JA plays a suppressing
333 role in ROS wave propagation.

334 Antagonistic interactions between SA and JA were previously reported in many studies (*e.g.,*
335 Spoel et al., 2003; Tada et al., 2008; Zhou et al., 2015; Caarls et al., 2015; Withers and Dong,
336 2016; Chen et al., 2021). We previously observed that when a combination of HS and HL was
337 applied to the same local Arabidopsis leaf, the ROS wave response originating from this leaf was
338 suppressed (Zandalinas et al., 2020a). Both HS and HL treatments resulted in the accumulation
339 of SA and JA in the local leaf, suggesting that this suppression could result from antagonistic
340 interactions between JA and SA (Zandalinas et al., 2020a). Indeed, we found that in the *aos1*
341 mutant the suppression of the ROS wave at the local leaf during the stress combination was
342 removed, supporting the hypothesis that SA and JA antagonize the function of each other, and
343 that JA might suppress the initiation of the ROS wave (Zandalinas et al., 2020a). In the current
344 work we clearly show that application of JA suppresses, and application of SA promotes, the
345 ROS wave in response to HL stress or wounding (Figure 4). Taken together, our results suggest
346 that in the *coi1* mutant the high basal levels of JA (Stotz et al., 2011; Supplementary Figure 2)

antagonize the function of SA, and that SA and JA have antagonistic functions in regulating the ROS wave (Figure 4, 5E). Of course, JA sensing could still play an important role in plant acclimation to HL stress and further studies are needed to address this question.

One protein, previously proposed to be at the core of SA-JA antagonistic interactions, is NPR1 (*e.g.,* Spoel et al., 2003; Tada et al., 2008; Zhou et al., 2015; Caarls et al., 2015; Withers and Dong, 2016; Chen et al., 2021). SA was shown to promote the monomerization of NPR1 via TRX-h3/5 that results in its nuclear localization and activation of transcriptional responses, while JA was shown to promote S-nitrosylation of NPR1 by S-nitrosoglutathione (GSNO) that keeps it as a multimer in the cytosol and prevents the activation of transcript expression (Caarls et al., 2015; Withers and Dong, 2016; Chen et al., 2021). NPR1 was further shown to be post-translationally regulated by ubiquitinoylation, SUMOylation and other post-translational modifications (Chen et al., 2021). Activation of transcriptional responses by SA was further shown to antagonize JA function and reverse its S-nitrosylation via GSNO Reductase (Caarls et al., 2015; Withers and Dong, 2016; Chen et al., 2021). Interestingly, in our hands, NPR1 was required for the systemic accumulation of ROS in response to a local treatment of HL stress or wounding, and for local and systemic acclimation to a local treatment of HL stress (Figure 3). We further show that in the absence of NPR1 (*npr1*) the expression of PR-1 is suppressed in local and systemic tissues of plants subjected to a local treatment of HL stress or wounding (Figure 3). Taken together, these findings suggest that NPR1 is required for SA to promote the ROS wave and trigger some of the transcripts required for plant acclimation to HL stress (Figure 5E). In this respect it should be noted that NPR1 was reported to play a key role as a master regulator of redox driven responses in the nuclei and to connect environmental cues with the circadian clock of plants (Zhou et al., 2015). Because the ROS wave is accompanied by a redox wave (Fichman and Mittler, 2021b), it could trigger different transcriptomic responses through NPR1 (and other transcriptional regulators such as MYB30; Fichman et al., 2020), that are modulated by an interplay between JA and SA (Figure 5E). ROS and redox signaling could therefore intersect with SA and JA signaling through NPR1 and control systemic accumulation of ROS and systemic plant acclimation to abiotic stresses or wounding (Figure 5E).

MATERIALS AND METHODS

Plant material, growth conditions, and stress treatments

Wild-type Arabidopsis (*Arabidopsis thaliana*; Col-0) and homozygous knockout mutants (Col-0 background) of *coi1* (SALK_095916C), *aos1-1* (SALK_017756C), *sid2* (SALK_093400C), *npr1* (SALK_204100C), *ein2* (CS3071), *etr1* (CS237), *aba2* (CS3835 and *aba2-11*), *max2* (SALK_028336C), and *max3* (SALK_023975C) were grown on peat pellets (Jiffy 7; Jiffy International, Kristiansand, Norway) for 4 weeks under controlled short-day light conditions of 10-h-light/14-h-dark, 50 µmol $m^{-2}s^{-1}$, and 21 °C room temperature. Homozygosity of each SALK line was determined via PCR (primers used are described in Supplemental Table S1). High light stress was applied using a ColdVision fiber optic LED light source (Schott, Southbridge, MA, USA) as previously described (Fichman et al., 2019; Fichman et al., 2022). Wounding stress was applied by puncturing a single leaf with 18 dressmaker pins simultaneously as described in Fichman et al., (2019).

Imaging of the ROS wave and hormone fumigation

ROS accumulation after administration of stress treatments was imaged and analyzed as previously described (Fichman et al., 2019). Plants were fumigated for 30 minutes in a glass aquarium using a nebulizer (Punasi Direct, Hong Kong, China) with solution containing 50 µM H_2DCFDA (Sigma-Aldrich, St. Louis, MO, USA) in 0.05M phosphate buffer, pH 7.4, and 0.01% (v/v) Silwet L-77 (LEHLE seeds, Round Rock, TX, USA). A single local leaf of the fumigated plant was treated with either high light or wounding stress as described above, and images of ROS accumulation were captured over the following 30 minutes using the IVIS Lumina S5 system (PerkinElmer, Waltham, MA, USA). Time course images of ROS accumulation were analyzed using the Living Image 4.7.2 software (PerkinElmer, Waltham, MA, USA). Measurement of total radiant efficiency in regions of interest (the local and systemic leaves) were used for data analysis as described in Fichman et al., (2019). Dye penetration controls were performed by fumigation and imaging with 0.3% (v/v) H_2O_2 for 10 minutes following 50 µM H_2DCFDA fumigation for 30 minutes (Fichman et al., 2019; Supplementary Figure 3). The whole-plant live ROS imaging method used in this study was validated in previous studies by measuring H_2O_2 in local and systemic tissues using the Amplex®-Red method as described below (Fichman et al., 2021, 2022; Also shown in Supplemental Figure 4). Hydrogen peroxide in

local and systemic leaves was quantified with Amplex®-Red (10-Acetyl-3,7-dihydroxyphenoxazine; ADHP; Thermo Fisher Scientific, Waltham, MA, USA). Leaves were flash frozen in liquid nitrogen, ground to powder, and resuspended in 50 µl 0.1M trichloroacetic acid (TCA; Thermo Fisher Scientific, Waltham, MA, USA). Following centrifugation for 15 min at 12,000 g, 4°C, the supernatant was buffered with 1 M phosphate buffer pH 7.4, and the pellet dried and used for dry weight calculation. H_2O_2 quantification at the supernatant was performed according to the MyQubit-Amplex®-Red Peroxide Assay manual (Thermo Fisher Scientific, Waltham, MA, USA), using an H_2O_2 calibration curve (Thermo Fisher Scientific, Waltham, MA, USA). Concentration values were normalized to dry weight of each sample (Fichman et al., 2022). Imaging of the ROS wave following administration of individual hormones was performed as described above with the addition of 20 µM jasmonic acid (Sigma-Aldrich, St. Louis, MO, USA) or 1 mM salicylic acid (Sigma-Aldrich, St. Louis, MO, USA) to the fumigation solution prior to stress treatment and imaging.

Systemic acquired acclimation following HL stress

Damage caused by HL stress was measured as previously described (Zandalinas et al., 2019; Fichman et al., 2020). High light stress (2000 µmol photons m^{-2} s^{-1}) was applied to either a local or a systemic leaf of a plant for 45 minutes to serve as the HL damage control. Following HL treatment, the exposed leaf was immediately sampled and placed in a tube containing 10 mL of ddH_20 and moved to a gentle shaker for one hour. After one hour, the electrolytic leakage was measured for each sample (treated, untreated, local, or systemic) using a conductivity meter Oakton CON 700 (Thermo Fisher Scientific, Vernon Hills, IL, USA). The samples were then boiled for 20 minutes. The boiled samples were moved to a shaker for one hour and the electrolytic leakage was measured for a second time. This process was also performed for plants receiving no HL treatment (untreated controls) and for plants that received 10 minutes of HL stress followed by a 50-minute incubation period under controlled conditions prior to the 45 minutes of HL that allows for acclimation to occur. The percentage of electrolytic leakage in each sample was determined by dividing the pre-boiling measurement of electrolytic leakage by the post-boiling electrolytic leakage in each sample.

Transcript expression analysis

The transcriptional responses to each stress (HL or wounding) were analyzed in local and systemic leaves at 0-, 10-, and 20-minute timepoints after application of the stress treatment as described in Fichman et al., (2020). Local and systemic leaves, located at 137.5° angle from the locally treated leaf in the plant rosette, were sampled for analysis. Following stress application, plants were sampled at the different time points and RNA was isolated. RNA extraction and purification were performed using RNeasy kit (Qiagen, Hilden, Germany) as described by the manufacturer's instructions and complementary DNA was synthesized for reverse-transcription quantitative PCR (RT-qPCR; Primescript RT Reagent Kit, Takara Bio, Kusatsu, Japan). RT-qPCR analysis was performed for the gene *PR1* (AT2G14610) with iQ SYBR Green supermix (Bio-Rad Laboratories, Hercules, CA, USA) and the CFX Connect Real-Time PCR Detection System (Bio-Rad Laboratories, Hercules, CA, USA) as described in Fichman et al., (2020). The forward and reverse primer sequences used for the analysis of the *PR1* transcriptional response were CGAACACGTGCAATGGAGTT and CACTTTGGCACATCCGAGTCT, respectively. Relative gene expression ($2^{-\Delta\Delta CT}$) was quantified using *ELONGATION FACTOR 1A* as the internal control (GAGCCCAAGTTTTTGAAGA and TAAACTGTTCTTCCAAGCTCCA). The relative increase in gene expression following stress treatment in each sample is shown as the increase compared to the untreated local sample that was collected alongside the treated samples.

Hormone measurements

Hormone extraction and quantification were performed as previously described (Balfagón et al., 2019; Sinha et al., 2022). Chromatographic separation was conducted on a reverse-phase C18 column (Gravity, 50 × 2.1 mm, 1.8 µm particle size; Macherey-Nagel GmbH, Dueren, Germany) using a MeOH : H2O (both supplemented with 0.1% (v/v) acetic acid) gradient at a flow rate of 300 µl min^{-1}. Hormones were quantified with a TQS triple quadrupole mass spectrometer (Micromass, Manchester, UK). All data were acquired and processed using Mass Lynx v.4.1 software.

Statistical analysis

Two-way analysis of variance (ANOVA) followed by a Tukey post hoc test was conducted for statistical analysis. Letters represent a statistically significant difference of at least $p<0.05$. Results for each experiment are displayed as box-and-whisker plots, with the borders corresponding to the 25^{th} and 75^{th} percentiles of the data. Each data value is included as a point

within each box plot, with the horizontal line representing the median and 'X' corresponding to the mean. Data points for ROS imaging are depicted as the determined total radiant efficiency ([p/s] / [$\mu W/cm^2$]) calculated within a chosen region of interest (ROI). Data for systemic acquired acclimation experiments is displayed as the relative amount of electrolytic leakage (shown as percent of control, with untreated local or systemic tissue acting as the control).

Accession numbers

Sequence data from this article can be found in the GenBank/EMBL data libraries under accession numbers: *ABA2* - NM_104113.5; *AOS1* - NM_123629.4; *COI1* - NM_129552.4; *EIN2* - NM_120406.5; *ETR1* - NM_105305.4; *GLR 3.3* - NM_103438.3; *GLR 3.6* - NM_115007.4; *HPCA1* - NM_124354.3; *RBOHD* - NM_124165.3; *RBOHF* - NM_105079.3; *MAX2* - NM_129823.3; *MAX3* - NM_001337112.1; *NPR1* - NM_105102.3; *PR1* - NM_127025.3; *SID2* - NM_127025.3.

Supplemental data

Supplemental Figure S1. Basal levels of JA, SA, and ABA in wild type and the *coi1*, *aos1-1*, *sid2*, and *npr1-1* mutants.

Supplemental Figure S2. Strigolactones are not required for the triggering of the ROS wave in local and systemic tissues, or for plant acclimation to HL stress.

Supplemental Figure S3. Mutants deficient in hormone production or signaling responses show no deficiency in absorption of fluorescent dye via fumigation.

Supplemental Figure S4. H_2O_2 quantification in local and systemic leaves of wild type (Col-0), *npr1-1*, *coi1*, *aba2-4*, and *aos1-1*, untreated or subjected to a local treatment of HL stress or wounding.

Supplemental Table S1. Primers for genotyping via PCR.

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FIGURE LEGENDS

Figure 1. Abscisic acid is required for acclimation to high light stress and the initiation of the ROS wave following wounding. **(A)** *Arabidopsis* plants were subjected to a high light (HL) stress or wounding treatment applied to a single leaf (L, Local), and ROS accumulation was imaged, using H_2DCFDA, in whole plants. Representative time-lapse images of whole plant ROS accumulation in wild type and *aba2-11* plants are shown alongside box plots of combined data from all plants used for the analysis at the 0- and 30-min time points (L and systemic [S] leaves). **(B)** Similar to A, except for the *aba2-4* mutant. **(C)** Ion leakage measurements of L and S leaves in Col-0 and *aba2-11* plants following HL stress. Local and systemic leaves that were exposed to an extended period of HL stress with no pretreatment (highlight), pretreated with HL for a short period of time and allowed to incubate prior to extended light exposure (local acclimated, La; and systemic acclimated, Sa), and control plants receiving no pretreatment, were measured. **(D)** Similar to C, except for the *aba2-4* mutant. All experiments were repeated at least three times with three plants per repeat. Two-way analysis of variance (ANOVA) followed by the Tukey post hoc test was conducted for statistical analysis. Letters represent a statistically significant difference of at least $p<0.05$. Results for each experiment are displayed as box-and-whisker plots, with the borders corresponding to the 25^{th} and 75^{th} percentiles of the data. Each data value is included as a point within each box plot, with the horizontal line representing the median and 'X' corresponding to the mean. Whiskers represent 1.5 times the minimum and maximum of the mean (1.5 times of the interquartile range).

Figure 2. Jasmonic acid insensitive mutants fail to acclimate to high light stress or induce a ROS wave response following a local treatment of high light or wounding. **(A)** *Arabidopsis* plants were subjected to a high light (HL) stress or wounding treatment applied to a single leaf (L, Local), and ROS accumulation was imaged, using H_2DCFDA, in whole plants. Representative time-lapse images of whole plant ROS accumulation in wild type and *coi1* plants are shown alongside box plots of combined data from all plants used for the analysis at the 0- and 30-min

time points (L and systemic [S] leaves). **(B)** Similar to A, except for the *aos1-1* mutant. **(C)** Ion leakage measurements of L and S leaves in Col-0 and *coi1* plants following HL stress. Local and systemic leaves that were exposed to an extended period of HL stress with no pretreatment (highlight), pretreated with HL for a short period of time and allowed to incubate prior to extended light exposure (local acclimated, La; and systemic acclimated, Sa), and control plants receiving no pretreatment, were measured. **(D)** Similar to C, except for the *aos1-1* mutant. All experiments were repeated at least three times with three plants per repeat. Two-way analysis of variance (ANOVA) followed by the Tukey post hoc test was conducted for statistical analysis. Letters represent a statistically significant difference of at least $p<0.05$. Results for each experiment are displayed as box-and-whisker plots, with the borders corresponding to the 25th and 75th percentiles of the data. Each data value is included as a point within each box plot, with the horizontal line representing the median and 'X' corresponding to the mean. Whiskers represent 1.5 times the minimum and maximum of the mean (1.5 times of the interquartile range).

Figure 3. Salicylic acid mutants are deficient in acclimation to high light stress and the ROS wave is unable to propagate to the systemic tissues in the *npr1-1* mutant. **(A)** *Arabidopsis* plants were subjected to a high light (HL) stress or wounding treatment applied to a single leaf (L, Local), and ROS accumulation was imaged, using H_2DCFDA, in whole plants. Representative time-lapse images of whole plant ROS accumulation in wild type and *sid2* plants are shown alongside box plots of combined data from all plants used for the analysis at the 0- and 30-min time points (L and systemic [S] leaves). **(B)** Similar to A, except for the *npr1-1* mutant. **(C)** Ion leakage measurements of L and S leaves in Col-0 and *sid2* plants following HL stress. Local and systemic leaves that were exposed to an extended period of HL stress with no pretreatment (highlight), pretreated with HL for a short period of time and allowed to incubate prior to extended light exposure (local acclimated, La; and systemic acclimated, Sa), and control plants receiving no pretreatment, were measured. **(D)** Similar to C, except for the *npr1-1* mutant. **(E)** Reverse transcription quantitative polymerase chain reaction (RT-qPCR) analysis for *PR-1* steady-state transcript levels in local and systemic leaves of wild type (Col-0) and *npr1-1* plants following highlight or wounding of a single leaf. Transcript expression is represented as the relative quantity ($2^{-\Delta\Delta CT}$) compared to an internal control (elongation factor 1α) in unwounded local tissue of wild-type (time 0). All experiments were repeated at least three times with three

plants per repeat. Two-way analysis of variance (ANOVA) followed by the Tukey post hoc test was conducted for statistical analysis. Letters represent a statistically significant difference of at least $p<0.05$. Results for each experiment are displayed as box-and-whisker plots, with the borders corresponding to the 25^{th} and 75^{th} percentiles of the data. Each data value is included as a point within each box plot, with the horizontal line representing the median and 'X' corresponding to the mean. Whiskers represent 1.5 times the minimum and maximum of the mean (1.5 times of the interquartile range).

Figure 4. JA suppresses the ROS wave while SA augments it in response to either high light or wounding of a local tissue. **(A)** *Arabidopsis* plants were untreated or pretreated with jasmonic acid, subjected to wounding (Top) or a high light (HL) stress (Bottom) applied to a single leaf (L, Local), and ROS accumulation was imaged, using H_2DCFDA, in whole plants. Representative time-lapse images of whole plant ROS accumulation in treated and untreated wild type plants are shown alongside box plots of combined data from all plants used for the analysis at the 0- and 30-min time points (L and systemic [S] leaves). **(B)** Similar to A, except that plants were untreated or pretreated with salicylic acid before wounding treatments or HL stress were applied. **(C)** Similar to A (Top), except for the *aos1-1* mutant. **(D)** Similar to A (Bottom), except for the *aos1-1* mutant. All experiments were repeated at least three times with three plants per repeat. Two-way analysis of variance (ANOVA) followed by the Tukey post hoc test was conducted for statistical analysis. Letters represent a statistically significant difference of at least $p<0.05$. Results for each experiment are displayed as box-and-whisker plots, with the borders corresponding to the 25^{th} and 75^{th} percentiles of the data. Each data value is included as a point within each box plot, with the horizontal line representing the median and 'X' corresponding to the mean. Whiskers represent 1.5 times the minimum and maximum of the mean (1.5 times of the interquartile range).

Figure 5. Ethylene insensitive mutants are unable to mount a systemic ROS wave response following a local wounding treatment, and a model. **(A)** *Arabidopsis* plants were subjected to a high light (HL) stress or wounding treatments applied to a single leaf (L, Local), and ROS accumulation was imaged, using H_2DCFDA, in whole plants. Representative time-lapse images of whole plant ROS accumulation in wild type and *ein2* plants are shown alongside box plots of combined data from all plants used for the analysis at the 0- and 30-min time points (L and

systemic [S] leaves). **(B)** Similar to A, except for the *etr1-1* mutant. **(C)** Ion leakage measurements of L and S leaves in Col-0 and *ein2* plants following HL stress. Local and systemic leaves that were exposed to an extended period of HL stress with no pretreatment (highlight), pretreated with HL for a short period of time and allowed to incubate prior to extended light exposure (local acclimated, La; and systemic acclimated, Sa), and control plants receiving no pretreatment, were measured. **(D)** Similar to C, except for the *etr1-1* mutant. **(E)** A model depicting the interactions between different plant hormones, the ROS wave and plant acclimation. All experiments were repeated at least three times with three plants per repeat. Two-way analysis of variance (ANOVA) followed by the Tukey post hoc test was conducted for the statistical analysis. Letters represent a statistically significant difference of at least $p<0.05$. Results for each experiment are displayed as box-and-whisker plots, with the borders corresponding to the 25^{th} and 75^{th} percentiles of the data. Each data value is included as a point within each box plot, with the horizontal line representing the median and 'X' corresponding to the mean. Whiskers represent 1.5 times the minimum and maximum of the mean (1.5 times of the interquartile range). Abbreviations: ABA, abscisic acid; ET, ethylene; JA, jasmonic acid; NPR1, NONEXPRESSER OF PR GENES 1; ROS, reactive oxygen species; SA, salicylic acid.

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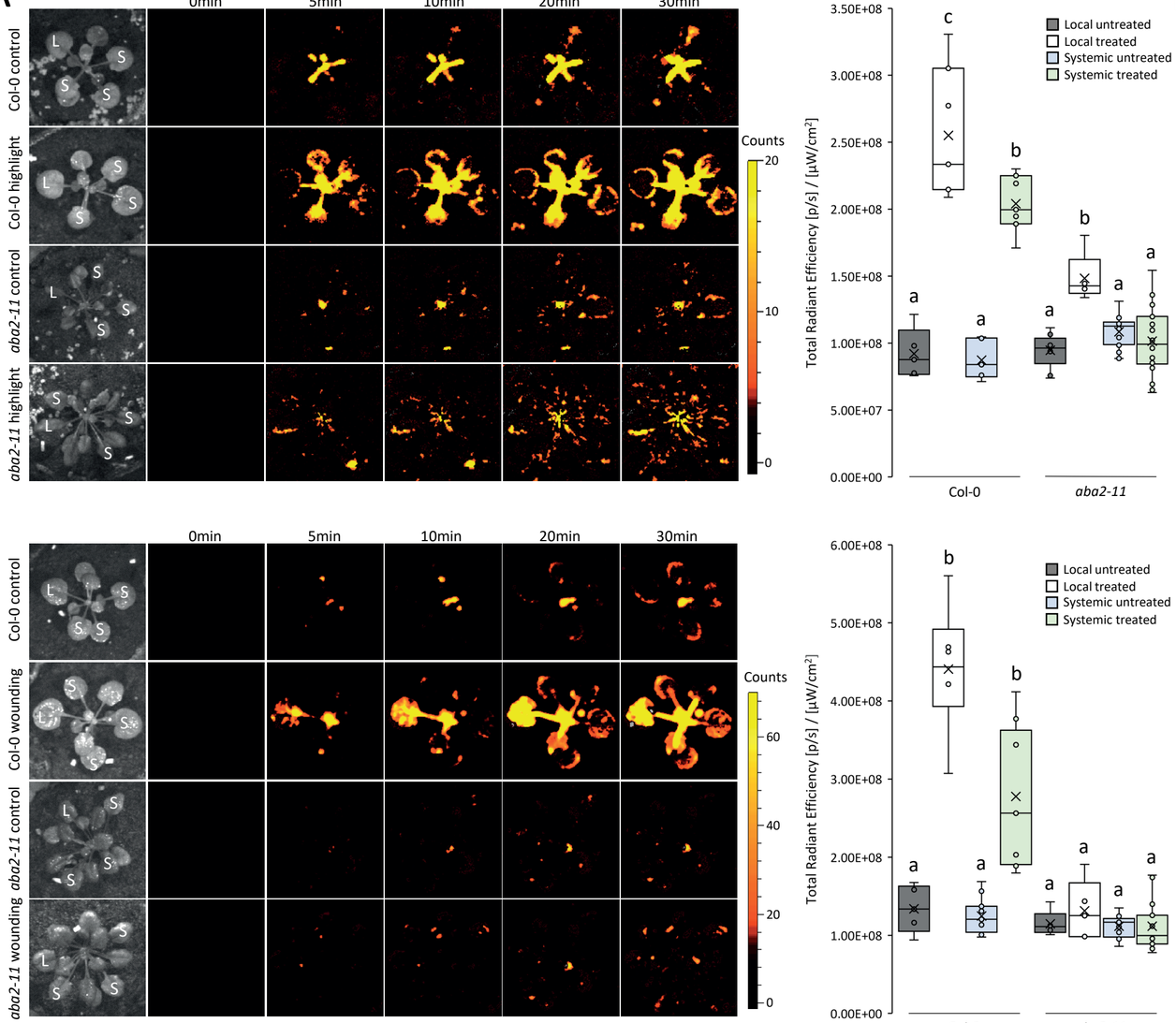
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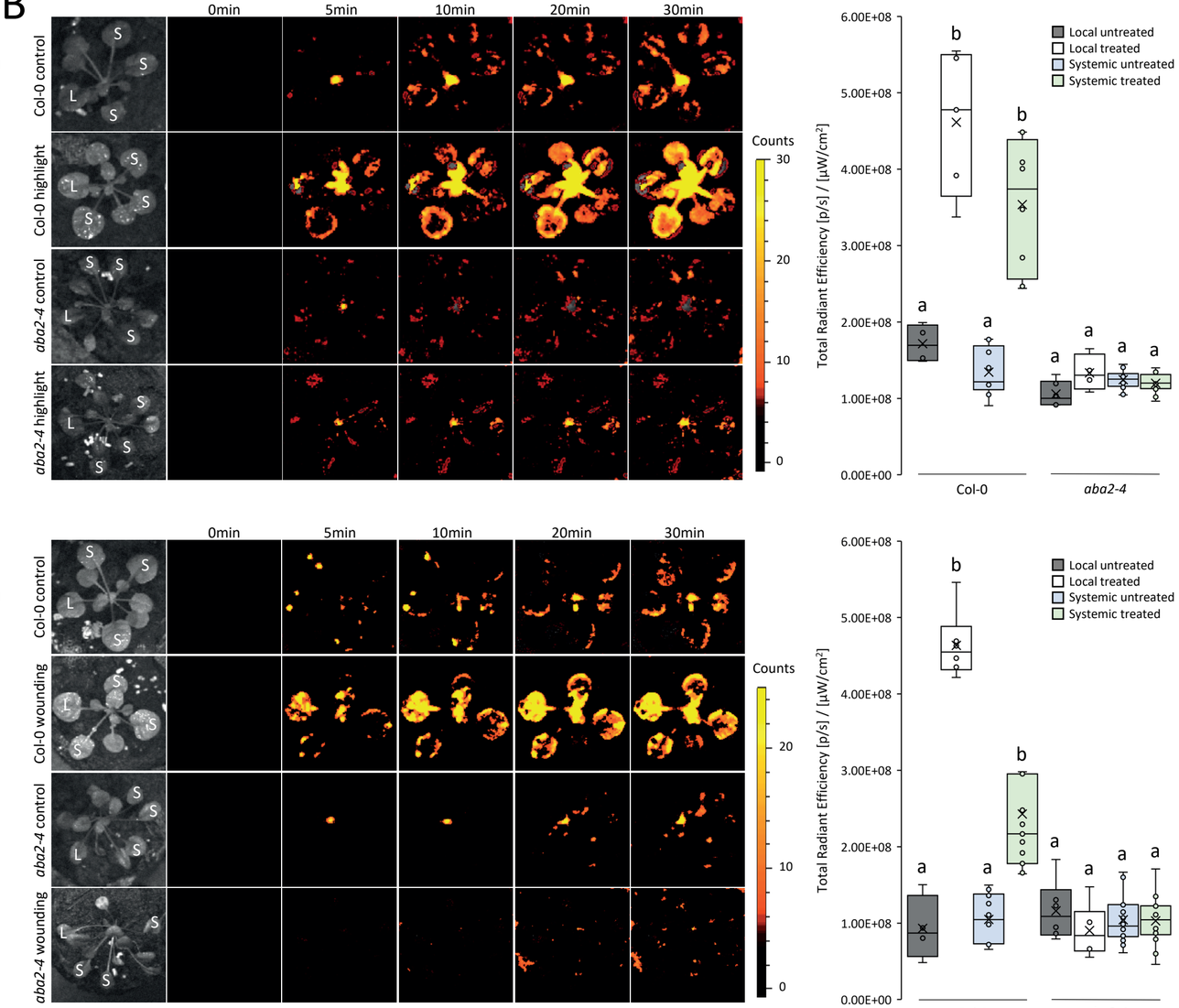
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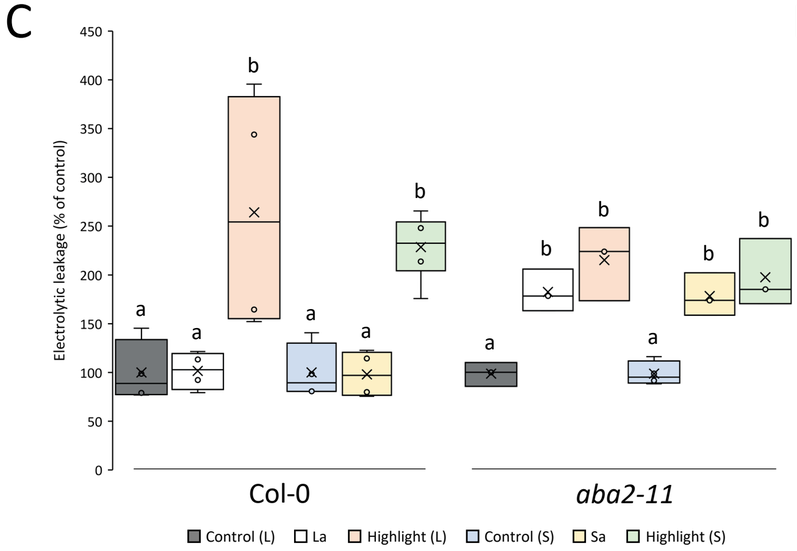
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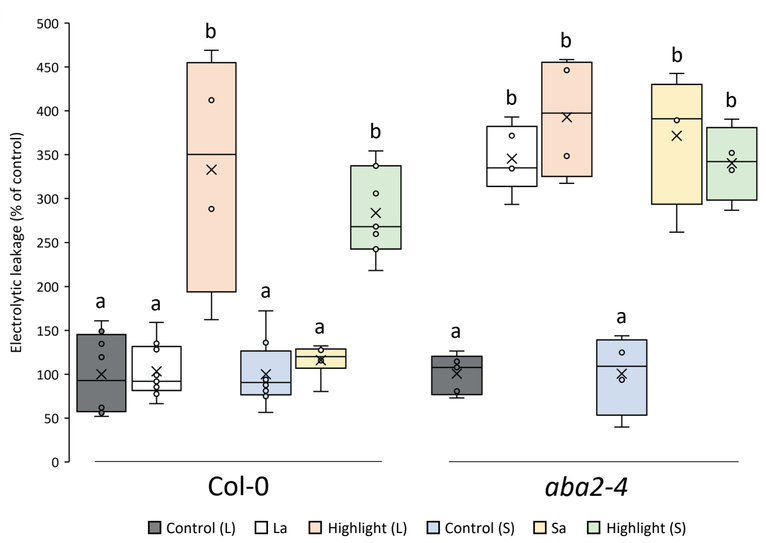
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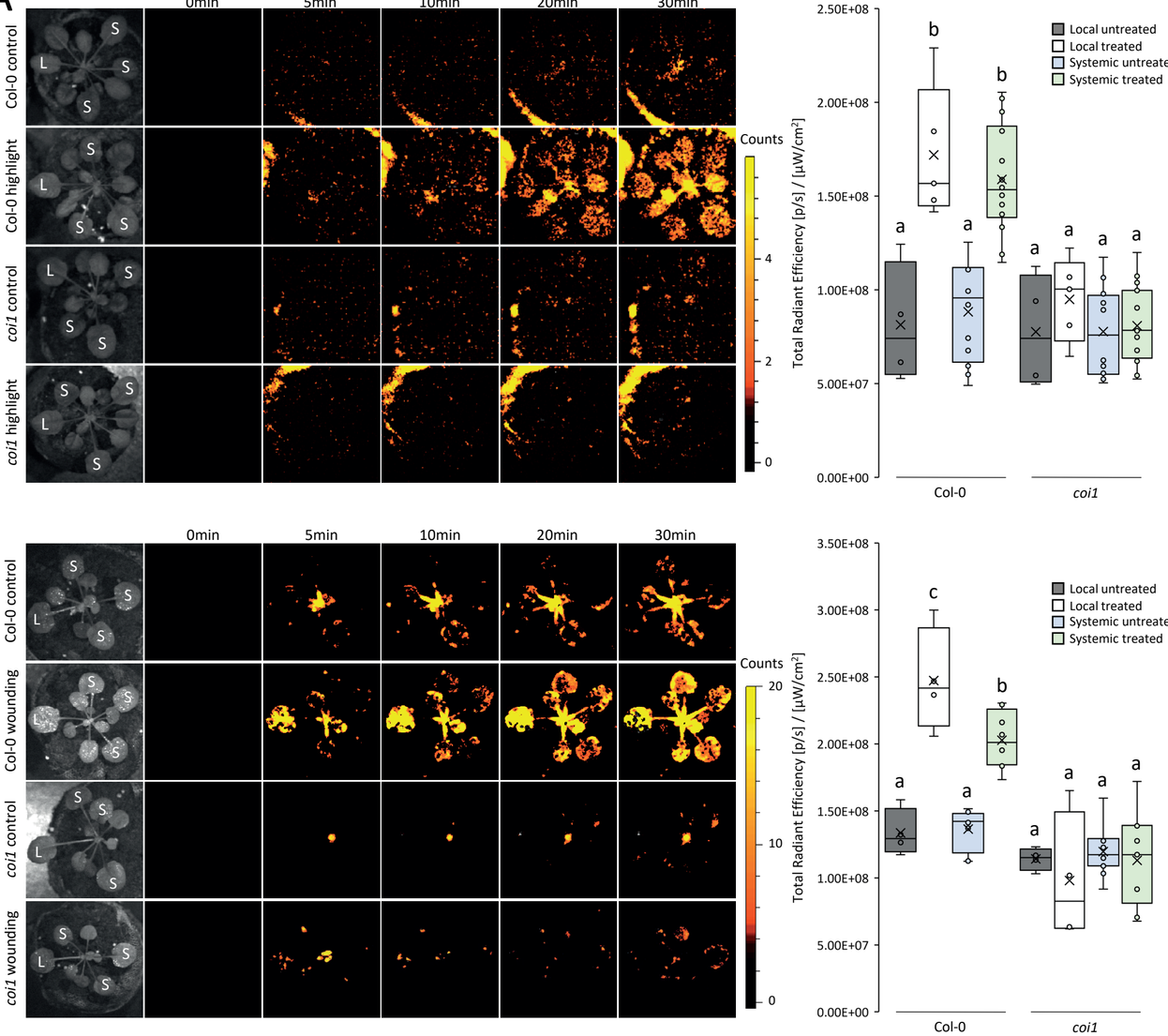
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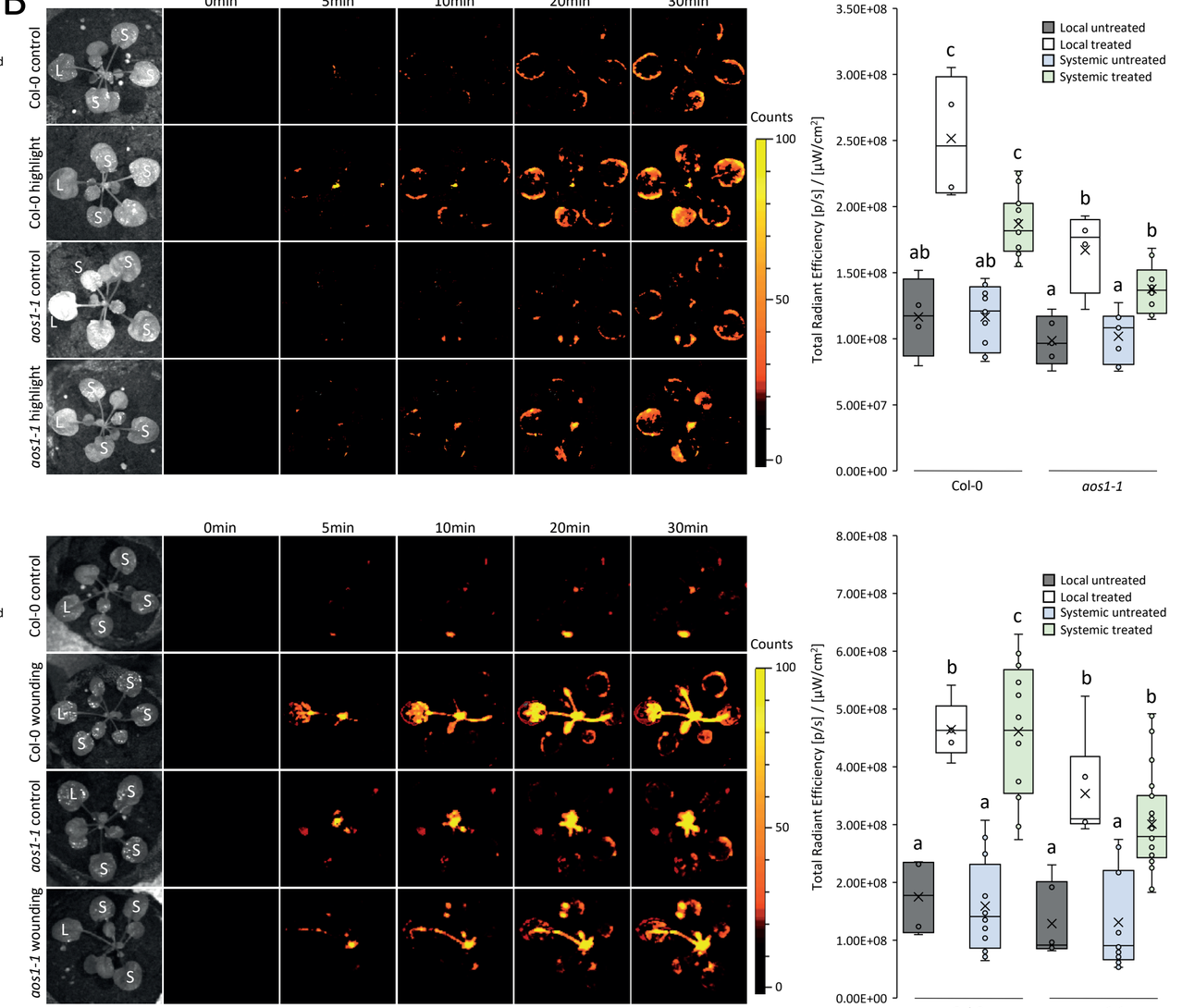
5min
10min
20min
30min
Col-0 control
Col-0 highlight
coi1 control
coi1 highlight
Counts
4
2
0
Total Radiant Efficiency [p/s] / [µW/cm²]
2.50E+08
2.00E+08
1.50E+08
1.00E+08
5.00E+07
0.00E+00
Local untreated
Local treated
Systemic untreated
Systemic treated
Col-0
coi1

B


0min
5min
10min
20min
30min
Col-0 control
Col-0 highlight
aos1-1 control
aos1-1 highlight
Counts
100
50
0
Total Radiant Efficiency [p/s] / [µW/cm²]
3.50E+08
3.00E+08
2.50E+08
2.00E+08
1.50E+08
1.00E+08
5.00E+07
0.00E+00
Local untreated
Local treated
Systemic untreated
Systemic treated
Col-0
aos1-1

0min
5min
10min
20min
30min
Col-0 control
Col-0 wounding
coi1 control
coi1 wounding
Counts
20
10
0
Total Radiant Efficiency [p/s] / [µW/cm²]
3.50E+08
3.00E+08
2.50E+08
2.00E+08
1.50E+08
1.00E+08
5.00E+07
0.00E+00
Local untreated
Local treated
Systemic untreated
Systemic treated
Col-0
coi1

0min
5min
10min
20min
30min
Col-0 control
Col-0 wounding
aos1-1 control
aos1-1 wounding
Counts
100
50
0
Total Radiant Efficiency [p/s] / [µW/cm²]
8.00E+08
7.00E+08
6.00E+08
5.00E+08
4.00E+08
3.00E+08
2.00E+08
1.00E+08
0.00E+00
Local untreated
Local treated
Systemic untreated
Systemic treated
Col-0
aos1-1

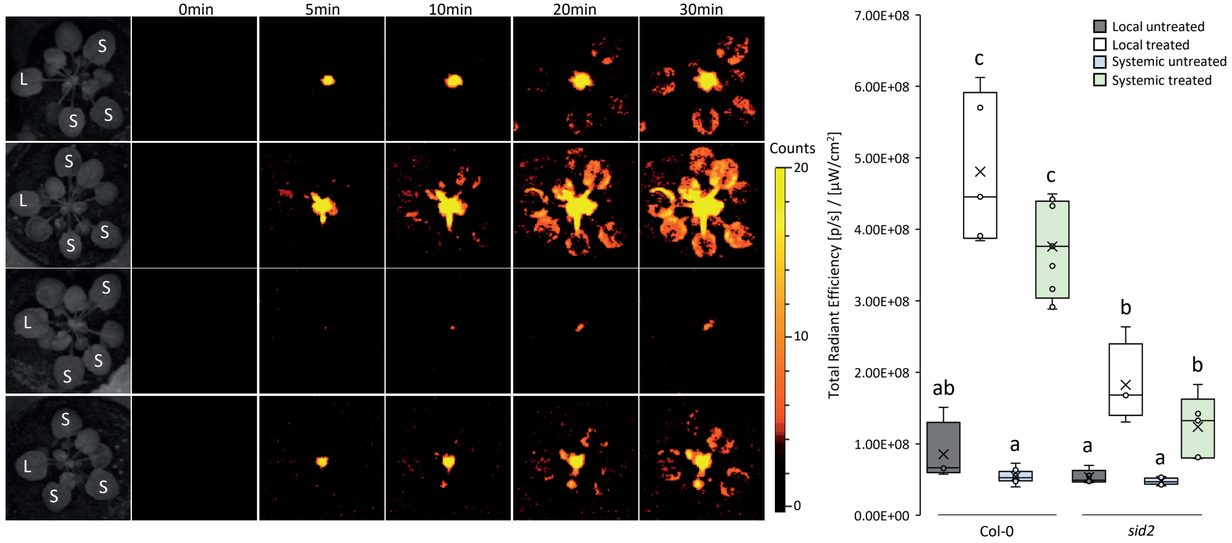
C

Electrolytic leakage (% of control)
500
450
400
350
300
250
200
150
100
50
0
Col-0
coi1
Control (L)
La
Highlight (L)
Control (S)
Sa
Highlight (S)

D

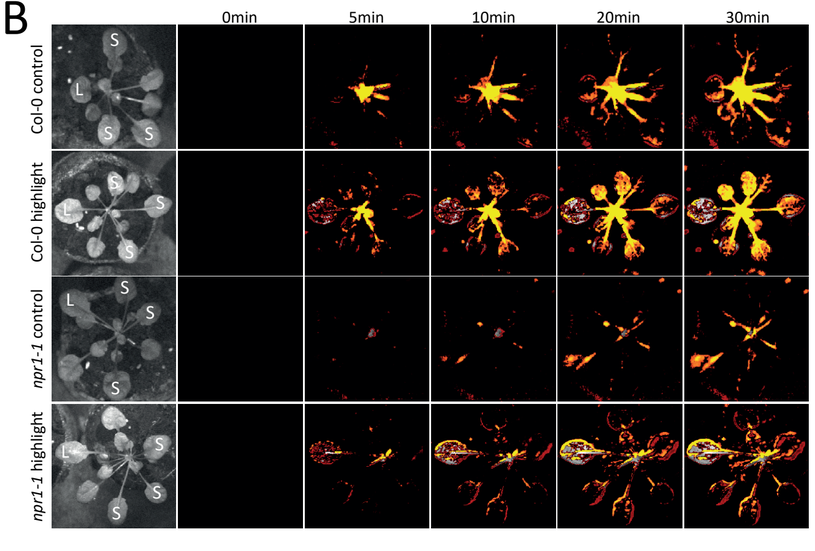
Electrolytic leakage (% of control)
500
450
400
350
300
250
200
150
100
50
0
Col-0
aos1-1
Control (L)
La
Highlight (L)
Control (S)
Sa
Highlight (S)

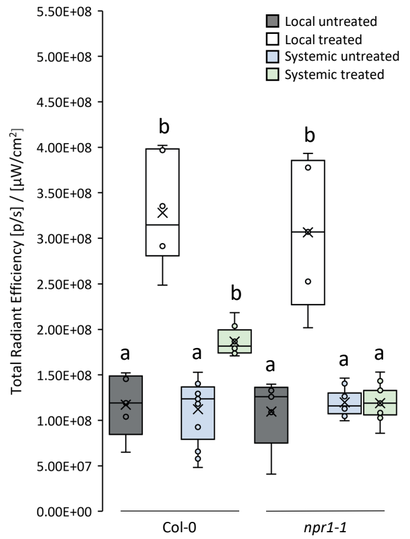
A

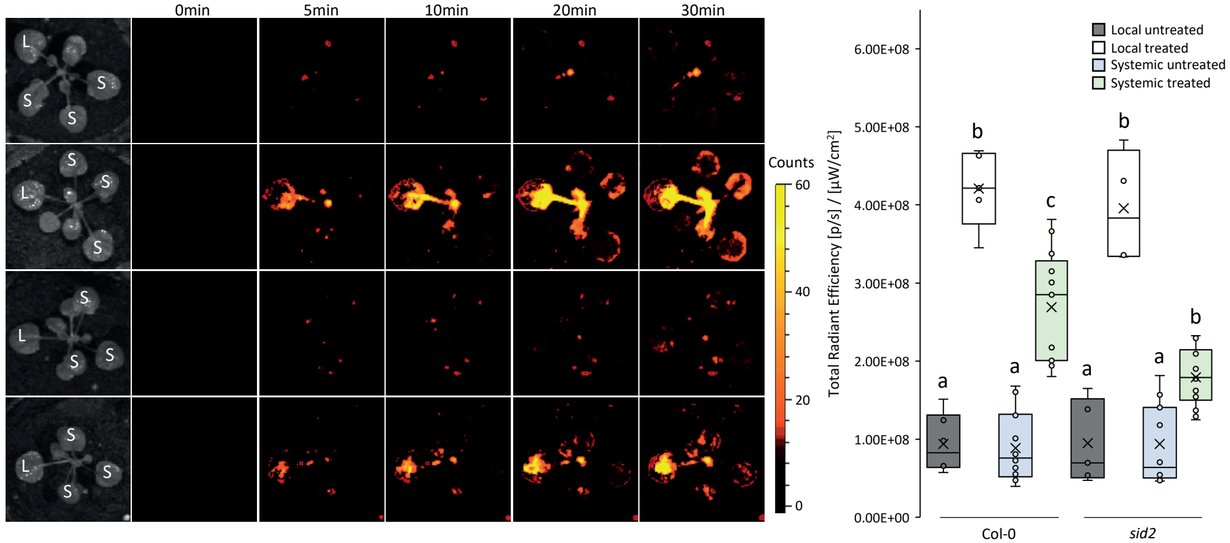

0min
5min
10min
20min
30min
Col-0 control
Col-0 highlight
sid2 control
sid2 highlight
Counts
20
10
0

Total Radiant Efficiency [p/s] / [µW/cm²]
7.00E+08
6.00E+08
5.00E+08
4.00E+08
3.00E+08
2.00E+08
1.00E+08
0.00E+00
Local untreated
Local treated
Systemic untreated
Systemic treated
c
c
b
b
ab
a
a
a
Col-0
sid2

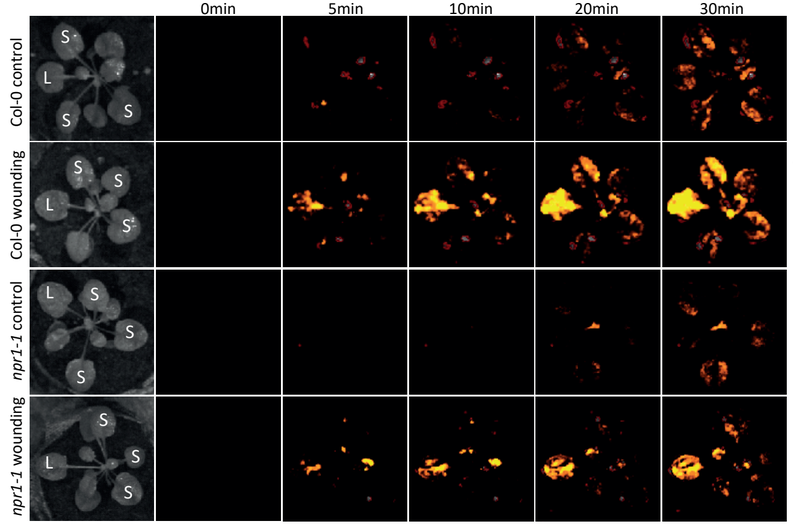
B

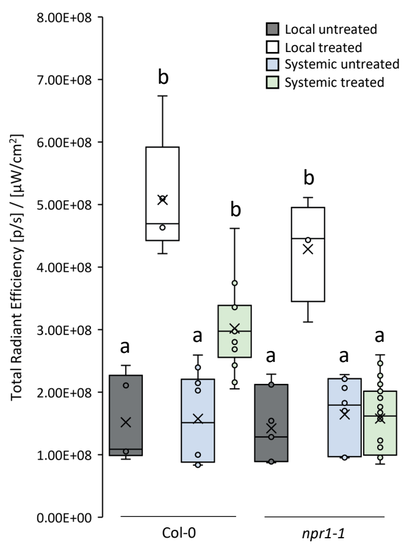

0min
5min
10min
20min
30min
Col-0 control
Col-0 highlight
npr1-1 control
npr1-1 highlight
Counts
30
20
10
0


Total Radiant Efficiency [p/s] / [µW/cm²]
5.50E+08
5.00E+08
4.50E+08
4.00E+08
3.50E+08
3.00E+08
2.50E+08
2.00E+08
1.50E+08
1.00E+08
5.00E+07
0.00E+00
Local untreated
Local treated
Systemic untreated
Systemic treated
b
b
b
a
a
a
a
a
a
Col-0
npr1-1

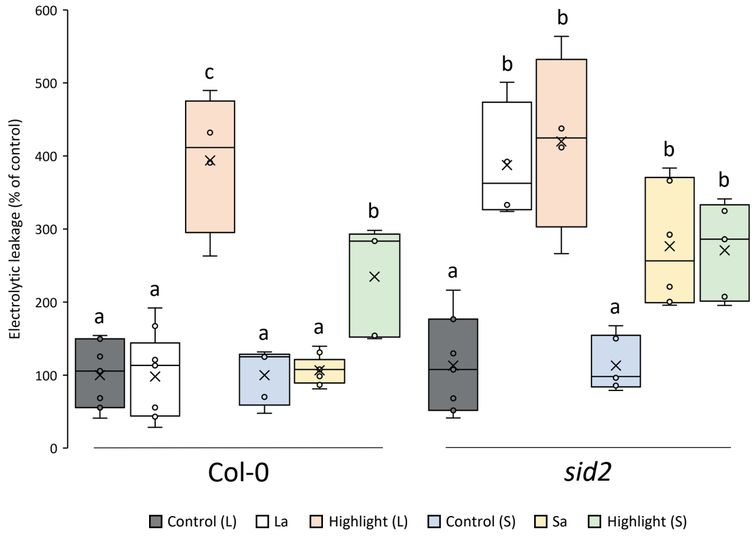

0min
5min
10min
20min
30min
Col-0 control
Col-0 wounding
sid2 control
sid2 wounding
Counts
60
40
20
0

Total Radiant Efficiency [p/s] / [µW/cm²]
6.00E+08
5.00E+08
4.00E+08
3.00E+08
2.00E+08
1.00E+08
0.00E+00
Local untreated
Local treated
Systemic untreated
Systemic treated
b
c
b
b
a
a
a
a
Col-0
sid2

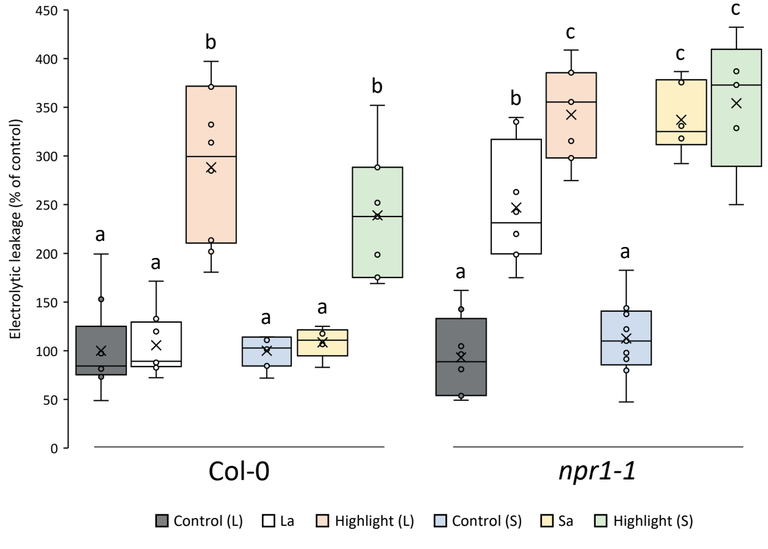

0min
5min
10min
20min
30min
Col-0 control
Col-0 wounding
npr1-1 control
npr1-1 wounding
Counts
30
20
10
0


Total Radiant Efficiency [p/s] / [µW/cm²]
8.00E+08
7.00E+08
6.00E+08
5.00E+08
4.00E+08
3.00E+08
2.00E+08
1.00E+08
0.00E+00
Local untreated
Local treated
Systemic untreated
Systemic treated
b
b
b
a
a
a
a
a
a
Col-0
npr1-1

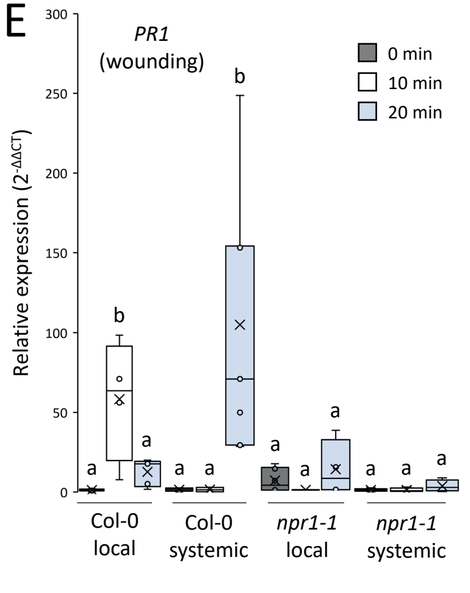
C

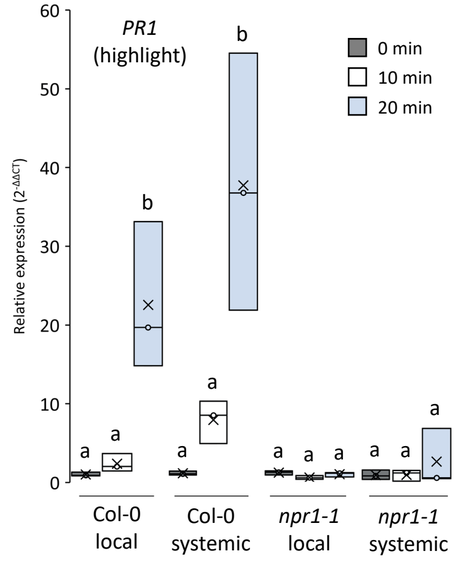

Electrolytic leakage (% of control)
600
500
400
300
200
100
0
c
b
b
b
a
a
a
a
a
b
Col-0
sid2
Control (L)
La
Highlight (L)
Control (S)
Sa
Highlight (S)

D

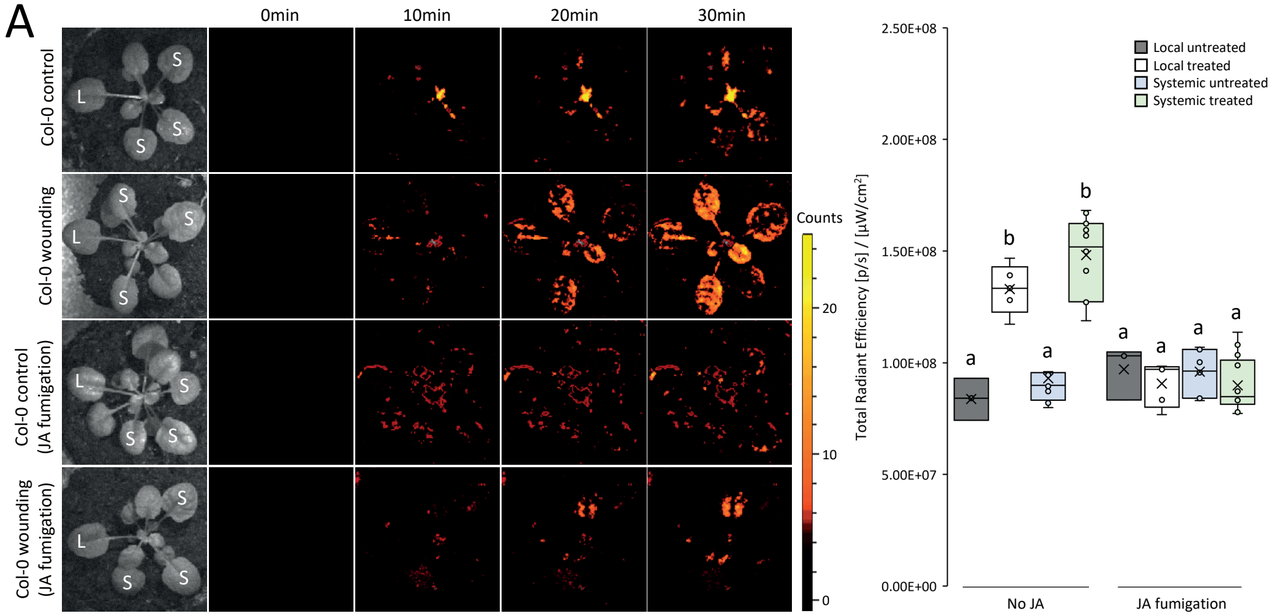

Electrolytic leakage (% of control)
450
400
350
300
250
200
150
100
50
0
b
b
c
b
b
c
c
a
a
a
a
a
a
Col-0
npr1-1
Control (L)
La
Highlight (L)
Control (S)
Sa
Highlight (S)

E


PR1
(wounding)
Relative expression (2-ΔΔCT)
300
250
200
150
100
50
0
0 min
10 min
20 min
b
b
a
a
a
a
a
a
a
a
a
Col-0
local
Col-0
systemic
npr1-1
local
npr1-1
systemic

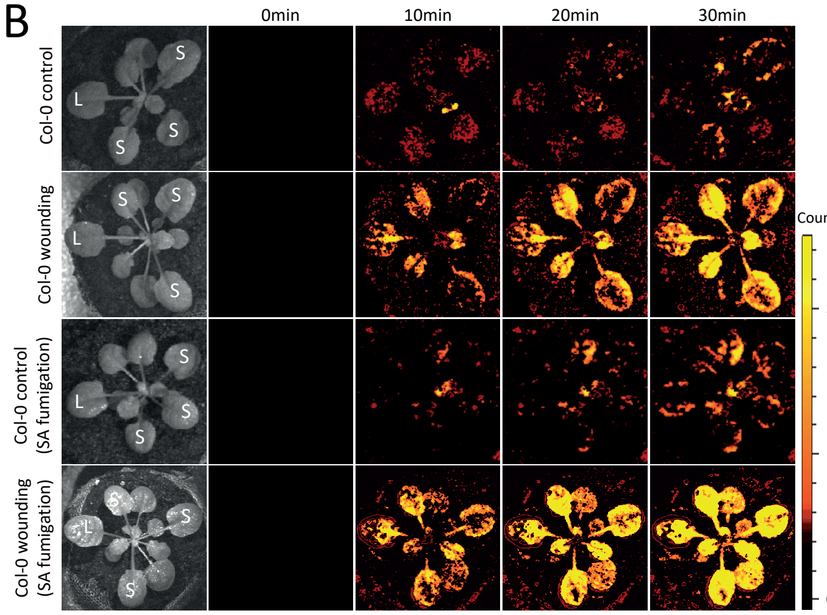

PR1
(highlight)
Relative expression (2-ΔΔCT)
60
50
40
30
20
10
0
0 min
10 min
20 min
b
b
a
a
a
a
a
a
a
a
a
Col-0
local
Col-0
systemic
npr1-1
local
npr1-1
systemic

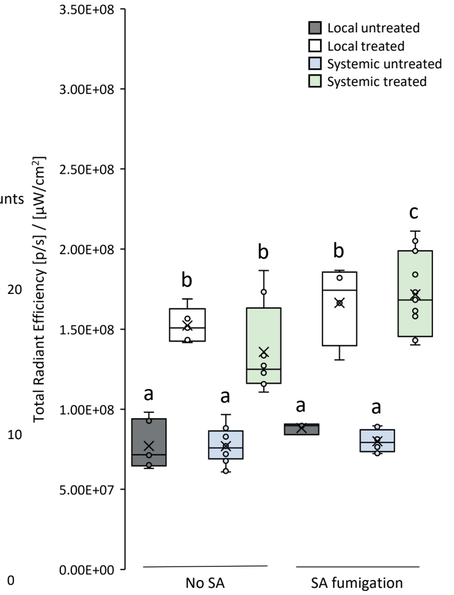
A

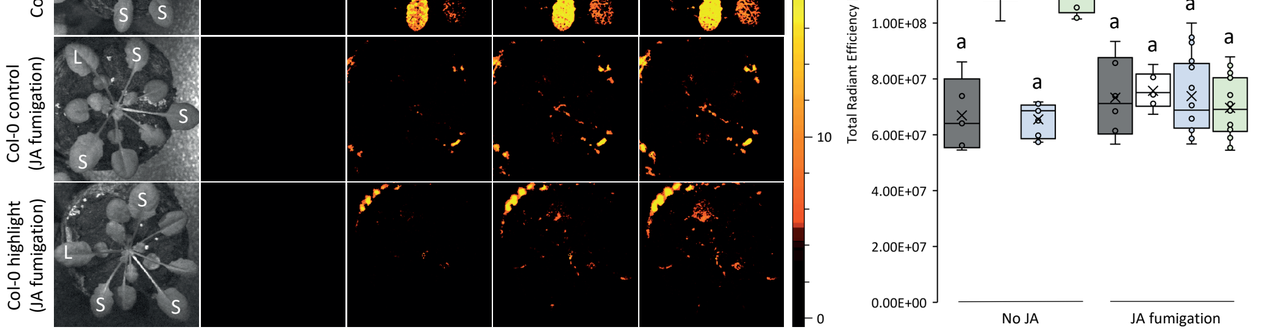

0min
10min
20min
30min
Col-0 control
Col-0 wounding
Col-0 control (JA fumigation)
Col-0 wounding (JA fumigation)
Counts
20
10
0

Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
2.50E+08
2.00E+08
1.50E+08
1.00E+08
5.00E+07
0.00E+00
a
b
a
b
a
a
a
a
No JA
JA fumigation

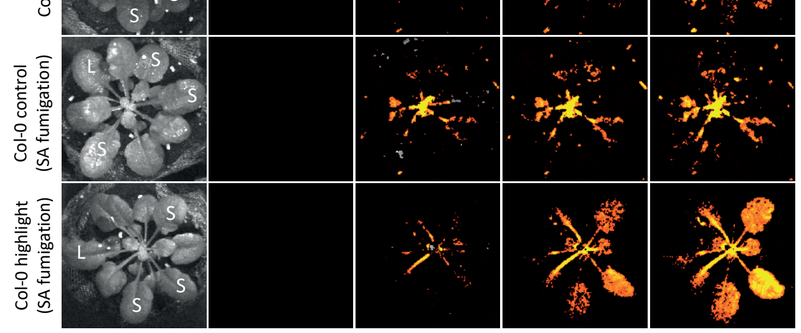
B

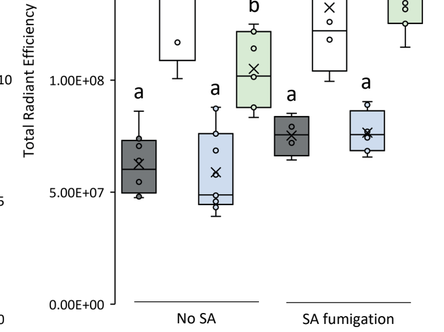

0min
10min
20min
30min
Col-0 control
Col-0 wounding
Col-0 control (SA fumigation)
Col-0 wounding (SA fumigation)
Counts
20
10
0


Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
3.50E+08
3.00E+08
2.50E+08
2.00E+08
1.50E+08
1.00E+08
5.00E+07
0.00E+00
a
b
a
b
a
b
a
c
No SA
SA fumigation

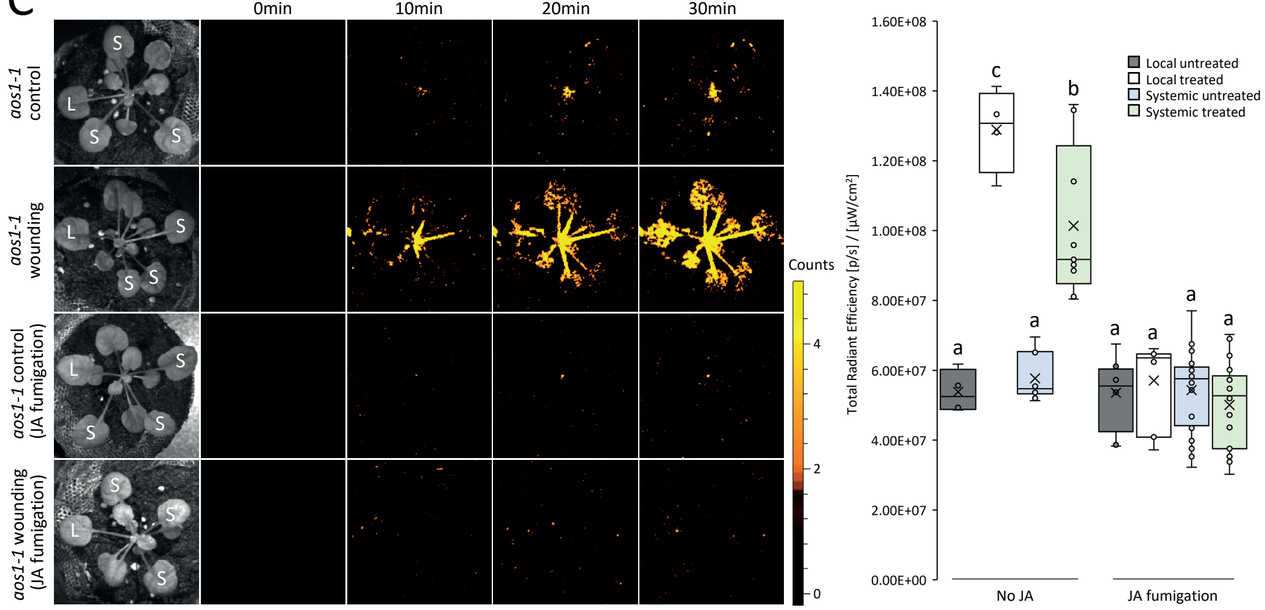

0min
10min
20min
30min
Col-0 control
Col-0 highlight
Col-0 control (JA fumigation)
Col-0 highlight (JA fumigation)
Counts
20
10
0

Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
2.00E+08
1.80E+08
1.60E+08
1.40E+08
1.20E+08
1.00E+08
8.00E+07
6.00E+07
4.00E+07
2.00E+07
0.00E+00
a
b
a
b
a
a
a
a
No JA
JA fumigation


0min
10min
20min
30min
Col-0 control
Col-0 highlight
Col-0 control (SA fumigation)
Col-0 highlight (SA fumigation)
Counts
15
10
5
0

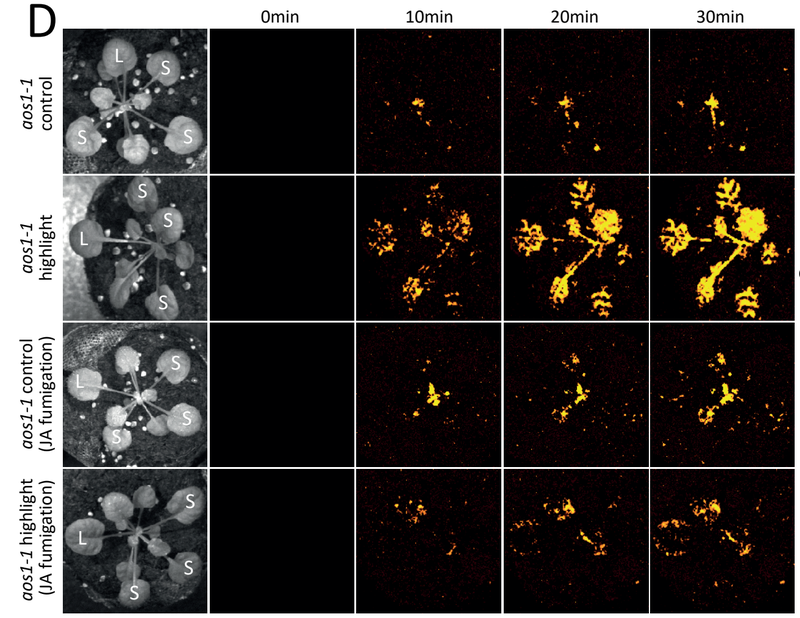

Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
2.50E+08
2.00E+08
1.50E+08
1.00E+08
5.00E+07
0.00E+00
a
b
a
b
a
b
a
c
No SA
SA fumigation

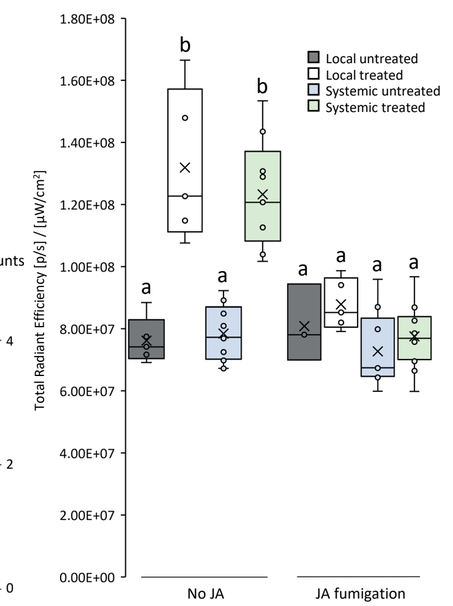
C


0min
10min
20min
30min
aos1-1 control
aos1-1 wounding
aos1-1 control (JA fumigation)
aos1-1 wounding (JA fumigation)
Counts
4
2
0

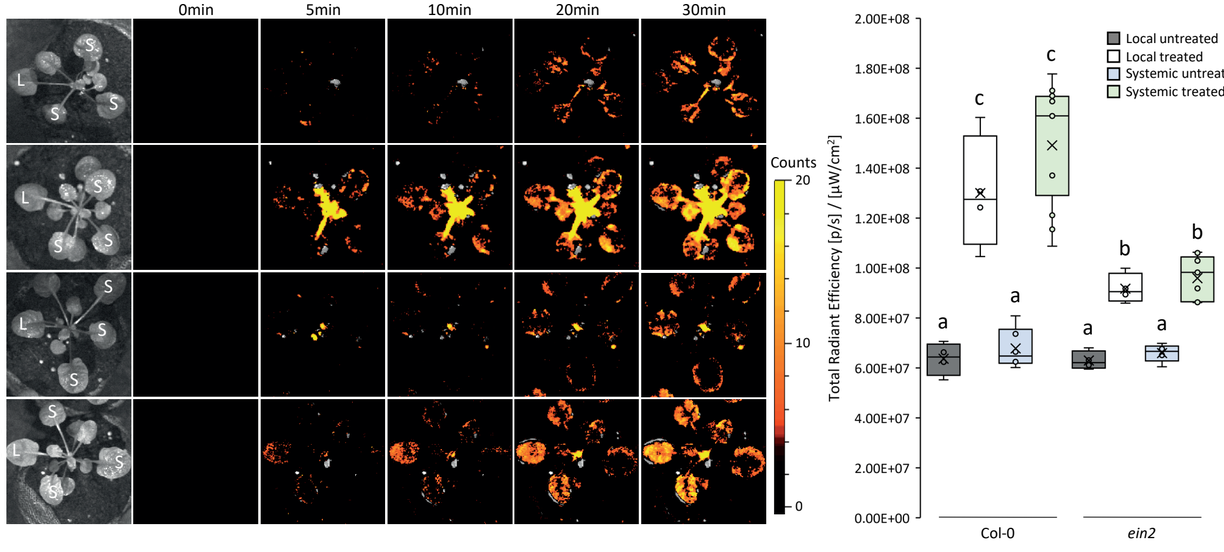
Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
1.60E+08
1.40E+08
1.20E+08
1.00E+08
8.00E+07
6.00E+07
4.00E+07
2.00E+07
0.00E+00
c
b
a
a
a
a
a
a
No JA
JA fumigation

D


0min
10min
20min
30min
aos1-1 control
aos1-1 highlight
aos1-1 control (JA fumigation)
aos1-1 highlight (JA fumigation)
Counts
4
2
0

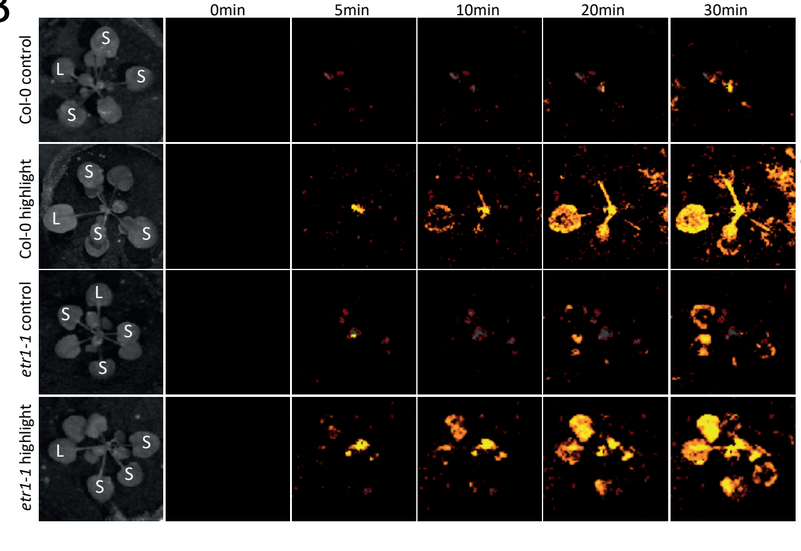

Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
1.80E+08
1.60E+08
1.40E+08
1.20E+08
1.00E+08
8.00E+07
6.00E+07
4.00E+07
2.00E+07
0.00E+00
b
b
a
a
a
a
a
a
No JA
JA fumigation

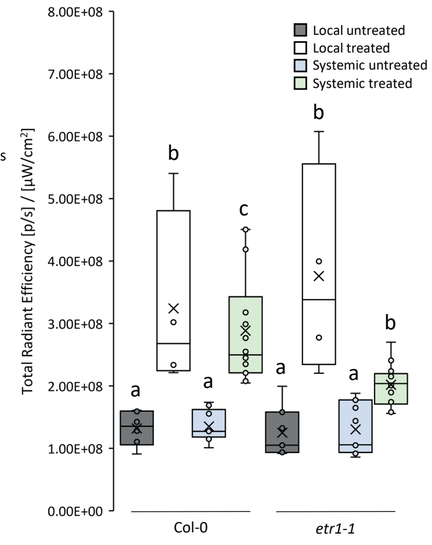
A

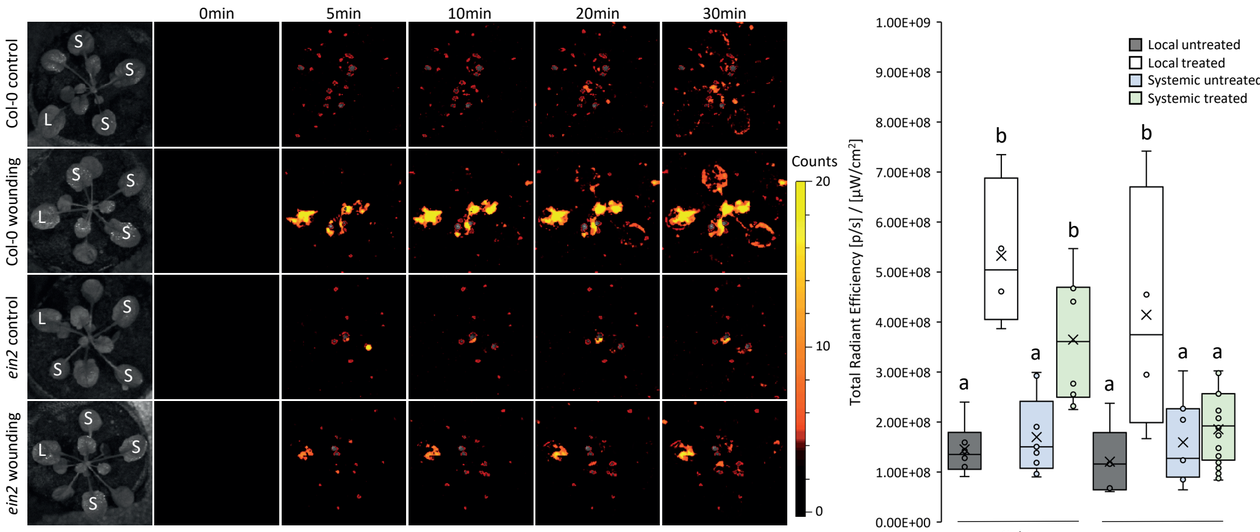

0min
5min
10min
20min
30min
Col-0 control
Col-0 highlight
ein2 control
ein2 highlight
Counts
20
10
0

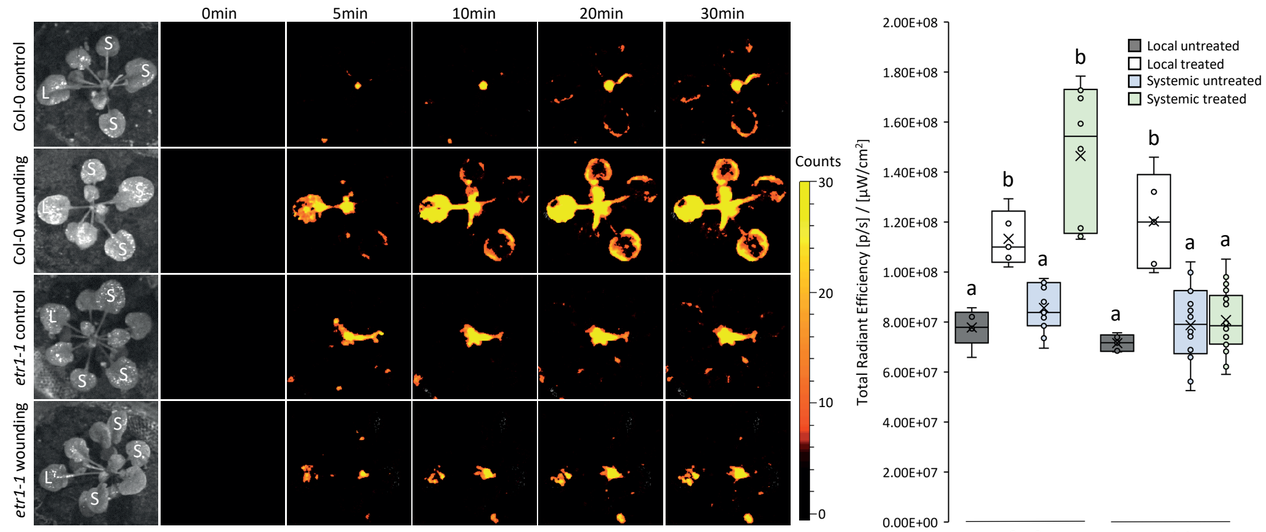
Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
2.00E+08
1.80E+08
1.60E+08
1.40E+08
1.20E+08
1.00E+08
8.00E+07
6.00E+07
4.00E+07
2.00E+07
0.00E+00
Col-0
ein2

B

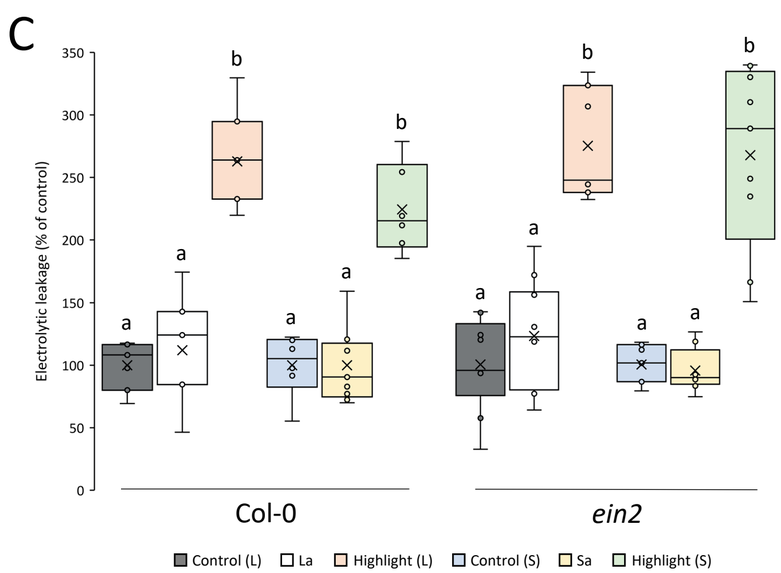

0min
5min
10min
20min
30min
Col-0 control
Col-0 highlight
etr1-1 control
etr1-1 highlight
Counts
20
10
5
0


Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
8.00E+08
7.00E+08
6.00E+08
5.00E+08
4.00E+08
3.00E+08
2.00E+08
1.00E+08
0.00E+00
Col-0
etr1-1

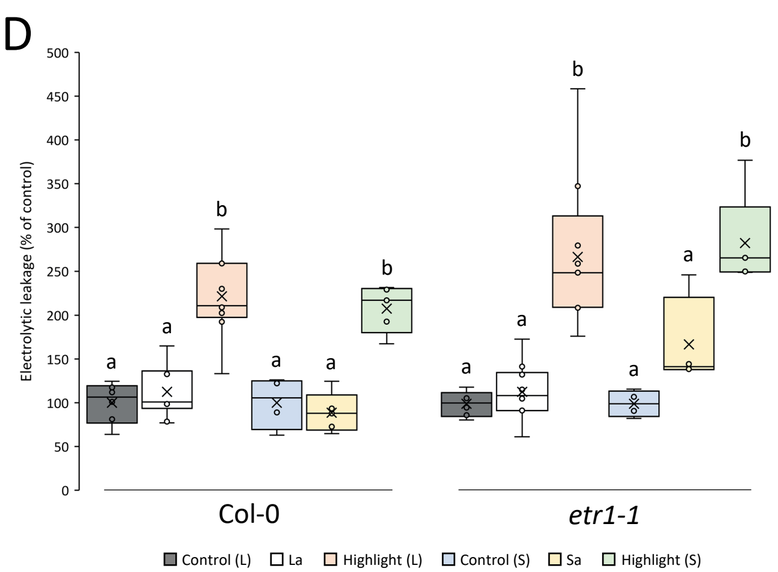

0min
5min
10min
20min
30min
Col-0 control
Col-0 wounding
ein2 control
ein2 wounding
Counts
20
10
0
Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
1.00E+09
9.00E+08
8.00E+08
7.00E+08
6.00E+08
5.00E+08
4.00E+08
3.00E+08
2.00E+08
1.00E+08
0.00E+00
Col-0
ein2


0min
5min
10min
20min
30min
Col-0 control
Col-0 wounding
etr1-1 control
etr1-1 wounding
Counts
30
20
10
0
Local untreated
Local treated
Systemic untreated
Systemic treated
Total Radiant Efficiency [p/s] / [µW/cm²]
2.00E+08
1.80E+08
1.60E+08
1.40E+08
1.20E+08
1.00E+08
8.00E+07
6.00E+07
4.00E+07
2.00E+07
0.00E+00
Col-0
etr1-1

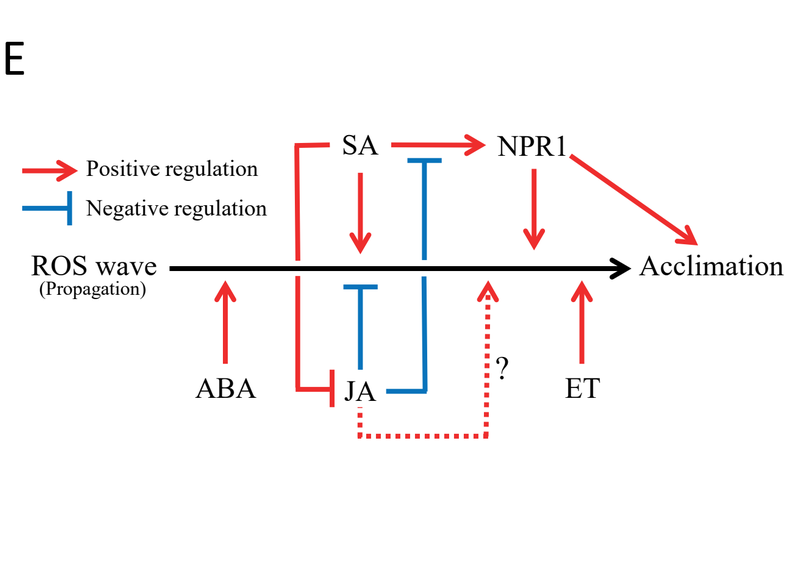
C


Electrolytic leakage (% of control)
350
300
250
200
150
100
50
0
Col-0
ein2
Control (L)
La
Highlight (L)
Control (S)
Sa
Highlight (S)

D


Electrolytic leakage (% of control)
500
450
400
350
300
250
200
150
100
50
0
Col-0
etr1-1
Control (L)
La
Highlight (L)
Control (S)
Sa
Highlight (S)

E


Positive regulation
Negative regulation
SA
NPR1
ROS wave
(Propagation)
Acclimation
ABA
JA
?
ET

Parsed Citations

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