The chloroplast plays a central role in facilitating MAMP-Triggered Immunity, pathogen suppression of immunity and crosstalk with abiotic stress.

Susan Breen[1], R. Muhammad Fraz Hussain[1], Emily Breeze[1], Hannah Brown[2], Ibrahim Alzwiy[3], Sara Abdelsayed[1], Trupti Gaikwad[3], and Murray Grant[1]

[1]University of Warwick School of Life Sciences
[2]United Kingdom Department of Health and Social Care
[3]University of Exeter College of Life and Environmental Sciences

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Abstract

Microbe associated molecular pattern (MAMP) triggered immunity research has traditionally centred around signal transduction pathways originating from activated membrane localised pattern recognition receptors (PRRs), culminating in nuclear transcription and post translational modifications. More recently, chloroplasts have emerged as key immune signalling hubs. Chloroplasts play a central role in integrating environmental signals. Notably MAMP recognition induces chloroplastic ROS (cROS) which is suppressed by pathogens effectors, which also modify the balance of defence hormone precursors, jasmonic acid (JA), salicylic acid (SA) and abscisic acid (ABA), whose precursors are chloroplast synthesised. This study focuses on how well characterised PRRs and co-receptors modulate chloroplast physiology, examining whether diverse signalling pathways converge to similarly modulate chloroplast function. Pre-treatment of receptor mutant plants with MAMP and D(Damage)AMP peptides usually protect against effector modulation of chlorophyll fluorescence and prevent *Pseudomonas syringae* effector mediated quenching of cROS and suppression of F_v/F_m. The MAMP-triggered immunity (MTI) co-receptor double mutant, *bak1-5/bkk1-1,* exhibits a remarkable decrease in F_v/F_m compared to control plants during infection, underlining the importance of MTI mediated signalling in chloroplast immunity. Further probing the role of the chloroplast in immunity we unexpectedly found that high light uncouples plant immune signalling.

Introduction

The plant immune system is multi-layered and complex. It traditionally comprises three modules; microbe associated molecular pattern (MAMP)-triggered immunity (MTI), effector-triggered immunity (ETI) and systemic acquired resistance (SAR) (Jones and Dangl, 2006; Shine*et al.* , 2019). The initial layer of defence, MTI, provides broad-spectrum defence against a diverse range of pathogens and has recently been shown to be involved in potentiating ETI responses, which can in turn reinforce MTI (Lu and Tsuda, 2021; Ngou *et al.* , 2021; Nguyen *et al.* , 2021; Yuan *et al.* , 2021). Classical pathogen cell surface receptors comprise transmembrane receptor-like kinases (RLKs) or receptor-like proteins (RLPs) including FLAGELLIN SENSING 2 (FLS2), EF-Tu RECEPTOR (EFR) and CHITIN ELICITOR RECEPTOR KINASE 1 (CERK1-2) which, respectively, detect flagellin and elongation factor thermo-unstable (EF-Tu) from bacterial pathogens and chitin from fungi (Yu *et al.* , 2017). However, an increasing number of MAMPs associated with a diverse range of pathogens have been identified (Noman, Aqeel and Lou, 2019). In addition, cell surface receptors can detect plant derived damage associated molecular patterns (DAMPs) found within extracellular spaces. Amongst DAMP receptors are the well-characterised RLKs, PEP RECEPTOR 1 (PEPR1) and PEPR2 which detect plant elicitor peptides, Peps. PEPR1, recognises Peps1-6 while PEPR2 recognises only Pep1

and Pep2 (Yamaguchi, Pearce and Ryan, 2006; Yamaguchi *et al.* , 2010). These Peps are cleaved from the C-terminus of plant PROPEPs during cell damage and the transcripts of PROPEP1-3 are induced by defence-related hormones methyl salicylate (MeSA) and methyl jasmonate (MeJA) (Huffaker, Pearce and Ryan, 2006; Yamaguchi *et al.* , 2010).

The pattern recognition receptors (PRRs), FLS2, EFR and PEPR1/2, are cell membrane localised and contain extracellular leucine rich repeat (LRR) surfaces where their ligands bind. Upon peptide detection by PRRs, co-receptors are recruited and bind to PRRs (and in some cases the ligand). The well characterised co-receptor Brassinosteroid Insensitive 1 (BRI1)-associated receptor kinase 1 (BAK1) belongs to the somatic embryogenesis receptor-like kinase family (SERK) which contains five members, one of which, SERK4/BKK1 (BAK1-LIKE 1), has high sequence similarity to BAK1 and has functional redundancy (He *et al.* , 2007). While BAK1 was first identified as a co-receptor for the Brassinosteroid receptor BRI1, involved in cell growth and division, it has become widely known for its role in plant immunity as plants containing the reduced function *bak1-5* allele have impaired FLS2, EFR and PEPR receptor function (Roux *et al.* , 2011; Schwessinger*et al.* , 2011). In contrast, *bkk1-1* still exhibits a reactive oxygen species (ROS) burst and MAP Kinase (MPK3, MPK4 and MPK6) activation, that is comparable to wild type plants, when treated with flg22 or elf18. However, the *bak1-5/bkk1-1* plants show minimal ROS and no MAPK (mitogen-activated protein kinase) activation in response to these PAMPs (Zipfel *et al.* , 2006; Roux *et al.* , 2011).

MTI triggers rapid calcium signalling, ROS and MAPK signalling cascades all of which involve plasma membrane to nuclear signalling (Noman, Aqeel and Lou, 2019). Microbes successful in colonisation secrete effectors to inter- or intracellular locations, which can dampen MTI signalling. Examples of such effector triggered suppression (ETS) include the AvrPto effector from *Pseudomonas syringae* which interacts with the PRRs FLS2 and EFR to dampen MTI in *Arabidopsis thaliana* (Xiang*et al.* , 2008) and AvrE from *P. syringae* and the maize pathogen *Pantoea stewartii* subsp. *Stewartia* which targets protein phosphatase 2A (PP2A) complexes in order to dampen MTI (Jin*et al.* , 2016).

Effectors collectively target an array of plant immune signalling components, many of which still remain elusive. Some effectors are directly or indirectly recognised by cytoplasmic receptors, most often belonging to the nucleotide-binding leucine-rich repeat receptors (NLRs) class, activating a second immune response, ETI (Jones and Dangl, 2006). There are three major classes of NLRs, the first two classically defined by their N-terminal; Toll-like, Interleukin-1 receptor domain TIR-NLRs (TNLs), coiled-coil domain CC-NLRs (CNLs). More recently the Resistance to Powdery Mildew 8 (RPW8) CC-NLR class (RNLs) (Jones, Vance and Dangl, 2016; Zhong and Cheng, 2016) have been described which act as "helper" NLRs for TNL and CNL "sensor" NLRs (Lu and Tsuda, 2021; Nguyen*et al.* , 2021; Maruta *et al.* , 2022). Interaction of an effector and NLR is usually associated with the macroscopic development of the hypersensitive response which restricts pathogen growth.

Classically, MTI research has centred around signal transduction pathways originating from the plasma membrane and activating nuclear transcription however, it is becoming increasingly recognised that chloroplasts are a key hub of immune signalling (Kachroo, Burch-Smith and Grant, 2021; Littlejohn *et al.* , 2021). Chloroplasts play a central role in integrating environmental signals and maintaining cellular homeostasis via retrograde signalling (de Souza, Wang and Dehesh, 2017; Breeze and Mullineaux, 2022). Relevant to host immune signalling, chloroplasts are also the site of chloroplastic ROS (cROS) generation and synthesis of defence hormone precursors, jasmonic acid (JA), salicylic acid (SA) and abscisic acid (ABA) (Littlejohn *et al.* , 2021). A key early MTI response is the rapid ROS generation, an apoplastic localised respiratory burst, primarily generated by RBOHD, a member of the nicotinamide adenine dinucleotide phosphate (NADPH) oxidase homologue (RBOH) family (Miller *et al.* , 2009). Activating MTI using an effector secretion deficient strain of *P. syringae*pv. *tomato* strain DC3000 (DC3000*hrpA*) also rapidly generates cROS production in *A. thaliana* , which is attenuated in the virulent DC3000 strain, shortly after effector delivery (de Torres Zabala *et al.* , 2015).

Concomitant with differences in cROS production during infection between the *P. syringae* strains DC3000 and DC3000*hrpA* , global transcriptome profiling of *A. thaliana* revealed significant alterations of nuclear

encoded chloroplast genes (*NECG* s). Remarkably, *NECGs,* represent ˜10% of all differentially upregulated genes and ˜30% of those significantly down regulated (de Torres Zabala *et al.* , 2015) during early MTI responses despite *NECGs* collectively account for only ˜14% of the transcriptome. Superimposed on this, effector delivery (2-3 hour post infection; hpi) caused transcriptional reprogramming of *NECGs* , suggesting ETS also targets *NECG*expression (de Torres Zabala *et al.* , 2015). These molecular signatures are reflected by physiological changes between DC3000 and DC3000*hrpA* challenge as evidenced by quantifying net photosynthetic CO_2 assimilation (A_{sat}) and chlorophyll fluorescence imaging parameters associated with electron transport during photosynthesis. DC3000 but not DC3000*hrpA*challenge induced a decrease in CO_2 assimilation, maximum dark-adapted quantum efficiency (F_v/F_m), maximum operating efficiency of photosystem II (PSII) (F_v'/F_m') and the efficiency with which light absorbed by PSII is used for quinone acceptor (QA) reduction and linear electron transport (F_q'/F_m') (de Torres Zabala*et al.* , 2015). In addition, DC3000 infection elicited an increase in Non-Photochemical Quenching (NPQ) and PSII redox state (qL; (F_q'/F_v')/(F_o'/F')) compared to DC3000*hrpA* (de Torres Zabala *et al.* , 2015). qL estimates the percentage of open PSII centres and the oxidation state of the primary PSII QA (Baker, 2008). An increase in qL suggests a decrease in electron transport from PSII. Thus, virulent pathogens can radically alter chloroplast physiological functions as part of their virulence strategy.

De novo induction of the plant hormone ABA during DC3000 infection contributes to ETS (de Torres-Zabala *et al.* , 2007) and was also recently shown to play a significant role in modulating chloroplast function. DC3000 induced suppression ofF_v/F_m was accelerated by co-infiltration of 10 µM ABA, effectively phenocopying DC3000 challenge of the *Arabidopsis* ABA hypersensitive protein phosphatase 2C (PP2C) *abi1/abi2/hab1* triple mutant. By contrast, the ABA deficient *Arabidopsis aldehyde oxidase 3* (*aao3*) mutant restricted DC3000 suppression ofF_v/F_m (de Torres Zabala *et al.* , 2015). Collectively these data show that the chloroplast is targeted early in pathogen infection and prior to bacterial multiplication, one of the earliest events being suppression of cROS.

This study focussed on how well characterised PRRs and co-receptors modulated chloroplast physiology, including accessing whether diverse signalling pathways converged to similarly modulate chloroplast function. Here we comprehensively examine chlorophyll fluorescence dynamics and the impact on attenuating chloroplast cROS. We show that pre-treatment of receptor mutant plants with MAMP and DAMP peptides generally offer protection against effector modulation of chlorophyll fluorescence but surprisingly, *fls2* plants pre-treated with chitin fail to provide such protection. The double mutant of the MTI co-receptors *bak1-5/bkk1-1* exhibits a remarkable decrease inF_v/F_m compared to control plants during infection, underlining the importance of MTI mediated signalling in underpinning chloroplast immunity. Expanding these findings to better understand the role of ABA and abiotic stress in chloroplast immunity we found that high light overrides the protection offered by MAMPs on wild-type plants.

Materials and Methods

Arabidopsis growth conditions. *Arabidopsis thaliana*seeds were sown in a compost mix comprising Levingston F2 compost + sand (LEV206):vermiculite (medium grade) mixed in a 6:1 ratio. Plants were grown in a controlled environment growth chamber under a 10 h day (21 °C; 120 µmol $m^{-2}s^{-1}$) and 14 h night (21 °C) with relative humidity of 65% for 5–6 weeks prior to use.

Arabidopsis peptide treatment. Pre-treatment of plants was conducted 16 h prior to bacterial challenge. Co-infiltration experiments were conducted by mixing the peptide or hormone of interest with the bacterial culture to attain the required final concentration and OD_{600} prior to infiltration. Concentrations of peptides or hormones were as follows; 1 µM of flg22, elf18, Pep1, Pep2 and Pep3, 100 µg/ml of Chitin (Sigma - C9752) and 10 or 100 µM ABA. H_2O was used as mock for pre-treatments.

Bacterial growth, maintenance and inoculation.*Pseudomonas syringae*strains were grown on solid Kings B media containing appropriate antibiotics as described (Truman, de Zabala and Grant, 2006). For inoculation, overnight cultures were grown with shaking (200 rpm) at 28 °C. Cells were harvested (2,500 g × 7 min), washed and re-suspended in 10 mM $MgCl_2$. Cell density was adjusted to OD_{600} 0.15 (0.75 x

10^8 colony forming units (cfu) ml^{-1}) for fluorescence imaging and confocal microscopy or OD_{600} 0.0002 for growth assays. All growth assays and ROS imaging experiments were performed at least three times. All fluorescence imaging experiments were performed at least four times.

Chlorophyll fluorescence imaging. Photosystem II chlorophyll fluorescence imaging of Arabidopsis rosettes was performed with a CF Imager (Technologica Ltd, Colchester, UK). **Normal light cycle;**plants were placed in the chamber for 40 min post-inoculation and then dark adapted for 20 min. This was followed by a saturating light pulse (6,349 μmol $m^{-2}s^{-1}$ for 0.8 s) to obtain maximum dark-adapted fluorescence (F_m). Actinic light (120 μmol $m^{-2}s^{-1}$ – the same as plant growth light intensity) was then applied for 15 min, followed by a saturating pulse to obtain maximum light adapted fluorescence ($F_{m'}$). The plants remained in actinic light for a further 24 min, then returning to a dark period of 20 min. This cycle (59 min duration) was repeated 23 times. **High light cycle;** plants were placed in the chamber for 40 min post-inoculation and then dark adapted for 20 min. This was followed by a saturating light pulse (6,349 μmol $m^{-2}s^{-1}$ for 0.8 s) to obtain maximum dark-adapted fluorescence (F_m). High light (650 μmol $m^{-2}s^{-1}$) was then applied for 15 min, followed by 3 saturating light pulses 5 minutes apart to obtain maximum light adapted fluorescence ($F_{m'}$). The plants remained in high light for a further 150 min then returned to a 20 min dark phase. This cycle (200 min duration) was repeated 8 times.F_m , $F_{m'}$ andF_o (minimal fluorescence with fully oxidized PSII centres) were used to calculate chlorophyll fluorescence parameters related to photosystem II: F_v/F_m(maximum dark-adapted quantum efficiency) and non-photochemical quenching (NPQ). These values were calculated as described by (Baker, 2008).

Bacterial growth measurements. Three leaves per plants (6 plants total) were syringe infiltrated with bacteria, OD_{600} 0.0002, and placed either under high light (450 μmol $m^{-2}s^{-1}$ or 600 μmol $m^{-2}s^{-1}$) or normal light (120 μmol $m^{-2}s^{-1}$) for 4 days. Three independent leaf discs per plant were excised and homogenised using a Tissue Lyser (Qiagen). Serial dilutions were spotted on Kings B media and colonies were counted 24 hpi.

Confocal microscopy. Col-0 plants were pre-treated with either water or peptide 16 h prior to bacterial challenge, then 3.5 hpi leaves were detached and floated, adaxial surface upwards, in a solution of 10 mM $MgCl_2$ containing 10 μM 2'7'-dichlorodihydrofluorescein diacetate (H_2DCF-DA; Enzo) for 40 min, then washed for 20 min in 10 mM $MgCl_2$ before imaging. Samples were mounted in perfluorodecalin (Littlejohn *et al.* , 2010) and images were captured on a Zeiss 880 using a 40× oil immersion lens. Argon laser excitation at 488 nm and an emission window of 512–527 nm was used to capture the dichlorofluorescein (DCF) signal. Chloroplast fluorescence was measured at 659–679 nm.

Results

MAMP pre-treatment protectsF_v/F_m suppression by *P. syringae* DC3000 infection.

Previous work showed that leaves pre-treated with flg22 24 hpi with virulent *P. syringae* DC3000 restricted effector induced suppression of maximum dark-adapted quantum efficiency (F_v/F_m) levels (de Torres Zabala*et al.* , 2015). To determine if this observation was true for other peptide elicitors, wild type, Col-0, *A. thaliana* leaves were pre-treated with the bacterial peptides flg22 (1 μM), elf18 (1 μM) and the fungal peptide chitin, (chi; 100 μg/ml) then 16 hpi with DC3000. All pre-treatments protected challenged leaves from DC3000 induced suppression of F_v/F_m over a 24 h period (Figure 1A). Figure 1B illustratesF_v/F_m images at 18 hpi showing pre-treated leaves (flg, elf, chi) have healthyF_v/F_m responses (red/orange leaves) whereas reduced F_v/F_m due to DC3000 infection following H_2O (mock) pre-treatment is characterised by their distinctive green/blue colour.

Flg22, elf18 and chitin are recognised by the plant cell surface pattern recognition receptors (PRR) FLS2, EFR and Cerk1-2, respectively. Elf18 pre-treatment of *fls2* leaves primed the plant and this cross-protection resulted in no change toF_v/F_m during DC3000 infection (de Torres Zabala *et al.* , 2015). These data indicate that activation of different MTI receptors can abrogate effector mediatedF_v/F_m suppression. Consistent with this hypothesis, flg22 pre-treatment on *efr1* (Figure 1C, D) or *cerk1-2* leaves (Figure 1E, F) results in protection against DC3000 mediated F_v/F_m suppression over a 24 h period. The level of protection offered by flg22

to*efr1* and *cerk1-2* mutants is comparable to the Col-0 control (Figure 1C, E). By contrast, pre-treatment of elf18 on*efr1* plants (Figure 1C, D) and chitin on *cerk1-2* plants (Figure 1E, F) failed to prevent suppression ofF_v/F_m following DC3000 infection. The F_v/F_m images at 18 hpi illustrate healthy (red/orange) flg22 pre-treated leaves on Col-0,*efr1* and *cerk1-2* plants compared to suppression ofF_v/F_m (green/blue) induced by DC3000 infection (Figure 1D, F) following H_2O treatment. In addition, elf18 pre-treatment protects *cerk1-2* plants from reduced F_v/F_m during DC3000 infection (Figure 2A, B). Notably, *cerk1-2* challenged leaves showed greater suppression of F_v/F_m compared to Col-0 (Figure 1D, 2A) indicating uncoupling chitin signalling may also weaken chloroplast immune responses. Surprisingly, while pre-treatment of *efr1* plants with chitin protected from DC3000 induced suppression of F_v/F_m levels chitin pre-treatment failed to protect *fls2* plants during DC3000 infection (Figure 2C-D), where DC3000 suppression ofF_v/F_m was indistinguishable from H_2O pre-treatment (Figure 2E-F).

MAMP pre-treatment compromises effector induced suppression of cROS.

The chloroplastic ROS (cROS) burst has emerged as an important component of plant immunity, as evidenced by early DC3000 effector delivery to attenuate the MTI induced cROS burst (de Torres Zabala *et al.* , 2015). Therefore, to assess the relationship betweenF_v/F_m and cROS production and thus the role of DC3000 effectors in this process, leaves were treated with the non-specific ROS reporter, 2'7'-dichlorodihydrofluorescein diacetate (H_2DCF-DA), to image cROS following DC3000 infection. Strong cROS induced following DC3000*hrpA* infection was evident 4.5 hpi whereas at this time cROS was minimal in DC3000 challenged leaves (Figure 3A, B). Notably, flg22 or elf18 pre-treatment of leaves prior to DC3000 infection showed cROS at 4.5 hpi (Figure 3C, D) indicating that the effectors secreted during DC3000 infection could not dampen cROS within a primed leaf.

bkk1-1/bak1-5 **plants show hyper suppression ofF_v/F_m during *P. syringae*DC3000 infection.**

As primary PRRs mediate chloroplast immune signals, the cell surface co-receptor mutants *bak1-5* , *bkk1-1* and double mutant*bkk1-1/bak1-5* were used to assess their contribution to alteredF_v/F_m dynamics during DC3000 infection. *bkk1-1* plants pre-treated with flg22, elf18 and chitin 16 h prior to infection with DC3000 showedF_v/F_m infection signatures equivalent to those measured following DC3000*hrpA* infection in Col-0 plants (Figure 4A-C, Sup Figure 1a, b). DC3000 challenged*bak1-5* plants showed a small but significantly greater suppression of F_v/F_m compared to Col-0 (Figure 4D), as expected given its partial loss of MTI function (Roux *et al.* , 2011). These data highlight both the power of quantitative chlorophyll fluorescence measurements and the ability to dynamically monitor effector impact on chloroplast physiology. Interestingly, F_v/F_m dynamics in DC3000*hrpA* challenged *bak1-5* leaves pre-treated with flg22 or elf18 prior to infection were WT in response, whereas chitin pre-treatment only partial protected againstF_v/F_m suppression in the*bak1-5* background (Figure 4D-F, Sup Figure 1c, d). Strikingly, MAMP pre-treatment with flg22, elf18 or chitin had no protective effect on F_v/F_m dynamics in the*bkk1-1/bak1-5* double mutant withF_v/F_m suppression being identical and often greater than the respective Col-0 control treatment (Figure 4E-G, Sup Figure 1d, e).

Pre-treatment of leaves with DAMPs results in protection of

F_v/F_m during *P. syringae* DC3000 infection.

Given the protection offered by MAMPs toF_v/F_m levels during DC3000 infection, we next tested whether similar protection was also conferred by plant derived Damage Associated Molecular Patterns (DAMPs). Using Pep elicitors, Col-0 leaves were first pre-treated with Pep1, Pep2 or Pep3 (all at 1 μM) 16 h prior to DC3000 challenge.F_v/F_m dynamics over 24 h revealed that Pep1 and 3 not Pep2 protected from DC3000F_v/F_m suppression (Figure 5A, B). Critically, Pep1 and 3 pre-treatment failed to alter DC3000F_v/F_m infection dynamics in the*pepR1-1x2-1* mutant (Yamaguchi *et al.* , 2010) (Figure 5C, D) whereas flg22 pre-treated *pepR1-1x2-1* leaves protected fromF_v/F_m suppression as seen above for Col-0 flg22 pre-treatment (Figure 5E, F). Interestingly, Pep1 and Pep3 pre-treatment only provided partial protection againstF_v/F_m suppression in DC3000 infected *fls2* leaves and no protection in *bkk1-1/bak1-5*plants (Sup Figure 2a-d). These data indicate a degree of cross protection of F_v/F_m between DAMPs and MAMP priming of the plant.

High light enhances F_v/F_m reduction during *P. syringae* DC3000 infection.

High light can result in photoinhibition and ROS accumulation around photosystem II. Excess light can be absorbed by the light-harvesting complexes and dissipated as heat via thermal energy dissipation (qE), linked to non-photochemical quenching mechanisms (NPQ; (Holt, Fleming and Niyogi, 2004)). High light also promotes perinuclear clustering of chloroplasts with genetic encoded H_2O_2 biosensors providing compelling evidence for cROS transport from the chloroplast stroma to the nucleus following high light exposure (Exposito-Rodriguez *et al.* , 2017). These data suggest that high light may pre-activate host immunity to DC3000 infection, therefore we examined F_v/F_m dynamics during DC3000 infection under standard (normal light) or high light conditions. A "normal" light (120 µmol $m^{-2}s^{-1}$) cycle of 1 h (as above), comprised 40 min of light before dark adaption, for 24 cycles. To ensure the duration of high light exposure would encompass early pathogen infection events, including expression of effector genes and assembly of the Type-III Secretion System (T3SS), we used a regime of 2.5 h high light (650 µmol $m^{-2}s^{-1}$) prior to dark adaption, enabling F_v/F_m measurements to be captured 8 times over a 26 h period. In comparison to DC3000 or DC3000*hrpA* challenge under normal light conditions (Figure 6A, B), high light resulted in a dramatic initial decrease of F_v/F_m within the first 6.5 hpi for both DC3000 and DC3000*hrpA* challenges. F_v/F_m in DC3000*hrpA* treated leaves partially recovered did not regain levels observed under normal light condition (Figure 6C, D). By contrast, leaves infected with DC3000 showed strong decreases in F_v/F_m over the entire 26 h. These were consistently significantly lower than that observed in infected leaves under normal light (Figure 6A-D). Interestingly, under high light conditions flg22 and elf18 pre-treatment failed to prevent the majority of the suppression of F_v/F_m and in fact show infection dynamics very similar to that observed in DC3000 challenged Col-0 leaves. These data imply high light was the dominant driver of F_v/F_m (Figure 6E, F). Consistent with this, high light treatment at the onset of infection even further suppressed the F_v/F_m infection dynamics observed for the *bkk1-1/bak1-5* plants (Figure 6G, H).

To ascertain the impact of high light induced F_v/F_m suppression we enumerated bacterial growth under high light and normal light conditions. As the strong F_v/F_m suppression under high light exhibited by DC3000 challenge is reminiscent of ETI responses (Littlejohn *et al.* , 2021) it was surprising that high light enhanced susceptibility (Figure 7, Supp Fig 3b). Interestingly, already hypersusceptible *bkk1-1/bak1-5* plants were even more susceptible to DC3000 infection under high light (450 µmol $m^{-2}s^{-1}$) suggesting that high light uncouples immunity through pathways independently of those guarded by classical MTI signalling (Figure 7). Interestingly, there was no significant difference in bacterial growth observed for *fls2* in comparison to Col-0 plants under high light (Figure 7). This apparent insensitivity of *fls2* plants to high light warrants further investigation. Notably, plants pre-adapted to high light were no more or less susceptible than plants exposed to high light immediately after DC3000 challenge (Figure 7 and Supp Figure 3). Despite showing the accumulation of anthocyanin compared to the cognate control plants under 120 µmol $m^{-2}s^{-1}$ (Supp Fig 3a), plants that had been acclimatised to high light treatment for 5 days showed similar enhanced susceptibility (Supp Fig 3b). This is despite significant accumulation of anthocyanin which are associated with accumulation of defensive metabolites (Gould, 2004; Schaefer and Rolshausen, 2006; Lev-Yadun and Gould, 2008). Thus, HL pre-adaptation is not required to elicit enhanced susceptibility, it is only required co-incident with pathogen infection to significantly enhance bacterial growth, and this is additional to that achieved by uncoupling classical MTI defences.

Pathogen induced suppression of ABA enhances F_v/F_m reduction.

ABA biosynthesis and signalling is hijacked by DC3000 to suppress immunity (de Torres-Zabala *et al.* , 2007; De Torres Zabala *et al.* , 2009). The impact of ABA mutants on virulence is reflected in F_v/F_m signatures (de Torres Zabala *et al.* , 2015). As ABA is made predominately in the chloroplasts, we investigated whether high light induced susceptibility was underpinned by ABA signalling. We monitored the impact of an ABA hypersusceptible signalling mutant (triple mutant) or the ABA insensitive biosynthetic mutant *Arabidopsis aldehyde oxidase 3* (*aao3*) on infection under normal and high light, monitoring both F_v/F_m and non-photochemical quenching (NPQ), the latter measuring the energy released as heat. The *aao3* mutant exhibited less suppression of F_v/F_m during DC3000 infection compared to Col-0 plants, reflected also in slightly

lower levels of NPQ in comparison to Col-0 (Figure 8A, B, Sup Fig 4a, b). By contrast the hypersensitive triple PP2C mutant (*abi1/abi2/hab1*) shows a faster decrease in F_v/F_m and a stronger increase in NPQ compared to Col-0 (Figure 8A, B, Supp Figure 4a, b). As previously reported (de Torres-Zabala *et al.* , 2007; De Torres Zabala *et al.* , 2009; Rubio *et al.* , 2009) under normal light conditions *aao3* plants are more resistant to DC3000 while the triple PP2C mutant is more susceptible (Figure 8C). However, under high light (450 µmol $m^{-2}s^{-1}$) Col-0 and *aao3* plants are more susceptible however there was no enhanced susceptibility evident in the triple PP2C mutant (Figure 8C) implying either ABA signalling is important for high light enhanced susceptibility or the *abi1/abi2/hab1* plants cannot support further bacterial multiplication. In addition, Col-0 plants grown under high light show accumulation of ABA after 5 days of high light growth and 9 days of high light with subsequent DC3000 infection compared to no increase in ABA under normal light or bacterial infection (Supp Figure 3c). In contrast the aao3 plants do not show an increase in ABA under normal or high light (Supp Figure 3c).

To next assess the interaction of ABA and light on chloroplast function during pathogen infection 10 µM ABA was co-infiltrated with DC3000 into Col-0 leaves. Under normal light, 10 µM ABA co-infiltration enhances the decrease in F_v/F_m levels as previously reported (de Torres Zabala *et al.* , 2015)(Figure 8D, E). Under high light conditions infiltration with 10 µM and 100 µM ABA treatments show a faster decrease ofF_v/F_m levels from 3.5 hours onwards (Figure 8F, G). To ensure that 10 and 100 µM ABA treatment was not toxic to *P. syringae* , DC3000 was plated on Kings B agar containing 0, 10, 50 and 100 µM ABA. Bacterial growth showed that there is a small reduction in growth of bacteria in the presence of ABA, notably being significant ($p<0.0005$)with increased ABA concentration, (Supp Figure 4c).

Discussion

We had previously shown that MTI significantly alters the expression of photosynthetic and nuclear encoded chloroplast localised genes (*NECGs*) within the first 2 h of challenge with the T3SS deficient non-pathogenic DC3000*hrpA* (de Torres Zabala *et al.* , 2015; Lewis *et al.* , 2015). Notably, this MTI response results in a strong suppression of *NECGs* yet does not significantly reduce maximum dark-adapted quantum efficiency (F_v/F_m) of PSII (de Torres Zabala*et al.* , 2015) compared to mock challenge. In comparison, virulent DC3000 can deliver effectors within 2-3 hpi, and strongly suppressesF_v/F_m as well as attenuating MTI induced cROS (de Torres Zabala *et al.* , 2015). Notably, DC3000 significantly reconfigures the expression of *NECG* within 3-4 h of infection (de Torres Zabala *et al.* , 2015), the timing of which coincides with the delivery of effectors into the plant cell.

Priming of plants to reduce bacterial colonisation has been previously demonstrated. Zipfel et al showed that *A. thaliana* Col-0 plants primed with flg22 or elf18 have reduced bacterial growth after infection with DC3000 compared to mock primed plants (Zipfel *et al.* , 2006). In addition, Wan et al showed that chitin pre-treatment also protects*A. thaliana* against DC3000 multiplication (Wan *et al.* , 2008). Thus, PRRs signal via a common pathway to induce MTI responses such as callose deposition, ROS and MAP Kinase activation. Activated MTI functions across pathogen classes, e.g. the fungal MAMP chitin can prime a plant against bacterial infection (Nühse *et al.* , 2000). Here we investigated the impact of such priming on the chloroplast as photosynthetic genes are significantly altered during disease and early immune signalling (Kachroo, Burch-Smith and Grant, 2021; Littlejohn*et al.* , 2021). Our data show that priming with flg22, elf18 or chitin fully attenuates suppression of the maximum dark-adapted quantum efficiency of PSII (F_v/F_m) by DC3000. Experiments with the broad-spectrum ROS and NOS (reactive nitrogen species) stain H_2DCF-DA show that this protection extends in part to restricting DC3000 suppression of cROS within primed leaves (Figure 3) (de Torres Zabala *et al.* , 2015). In general, plants that have lost a MAMP; FLS2, EFR, CERK1-2 or DAMP receptor, PepR1-1x2-1 can sustain normalF_v/F_m during bacterial infection by priming with an alternative M/DAMP i.e., *efr* , *cerk1-2*and *pepR1-1x2-1* plants retain normalF_v/F_m with flg22 pre-treatment (Figures 1, 2, 5E, F and 9). We did observe however that Pep1 and 3 provided reduced attenuation ofF_v/F_m suppression in *fls2*plants compared to Col-0 (Sup Fig 2) and unexpectedly, chitin failed to protect F_v/F_m suppression in*fls2* plants (Figure 2E, F and 9) whereas *efr* plants are protected by chitin treatment (Figure 2C, D and 9). These findings highlight specificity between initial downstream signalling through different

PRRs. These data suggest that the activation of immune signals transduced by PepR1/PepR2 and CERK1 are possibly not sufficiently strong to protect against bacterial infection in the absence of FLS2. Notably, in *efr* mutants pre-treated with chitin, FLS2 activation could over-ride those chloroplast processes targeted by bacteria during infection. These data suggest that there may be a requirement for pre-formed complexes with co-receptors to attenuate chloroplast immune priming.

PRRs represent the first line of induced defence and most require homo or heterodimerisation with a receptor for effective immune signalling. Chitin induces the dimerization and cross-linking of AtCERK1 which is required for immune signalling (Liu *et al.* , 2012). By comparison, FLS2 and EFR are known to interact with co-receptors BAK1 or BKK1, members of the SERK (SOMATIC EMBRYOGENESIS RECEPTOR KINASEs) protein family. Perception of flg22 or elf18 by their ligand leads to phosphorylation of their intracellular kinase domains and induction of downstream immune signals (Zhang and Zhou, 2010). BAK1 was originally identified as the co-receptor for the Brassinosteroid (BR) cell surface receptor BRI1. MTI is impaired in *bak1-5* in response to flg22, elf18 or Pep1, leading to a reduced ROS burst and dampened MAPK activation (Roux *et al.* , 2011). However, this mutant is not impaired in BR signalling (Schwessinger *et al.* , 2011). In contrast, the ROS burst and MAPK responses to flg22, elf18 or Pep1 elicitation in a loss of function *bkk1-1* mutant are similar to wild type (Roux *et al.* , 2011). Priming of either *bkk1-1* or *bak1-5* individual mutants with flg22 and elf18 shows no suppression of F_v/F_m indicating that these peptides can protect the PSII function from bacterial infection (Figure 4 and 9). This is comparable to the immune response functions observed for *bkk1-1* and the BR responses observed for *bak1-5* (Roux *et al.* , 2011; Schwessinger *et al.* , 2011). Chitin peptide priming prevented F_v/F_m suppression by DC3000 in *bkk1-1* plants but provided only partial protection in *bak1-5* plants (Sup Fig 1a-d, and Fig 9), consistent with the compromised immune signalling in *bak1-5* . The double mutant *bkk1-1/bak1-5* has dramatically reduced immune responses to flg22 and elf18 elicitation (Roux *et al.* , 2011). Here we demonstrated that at the level of chloroplast function, priming with flg22, elf18 or chitin offered no protection. Rather, we measure a quantitative hyper reduction in F_v/F_m in comparison to Col-0 plants (Figure 4 and 9, Sup Fig 1d, e). The fact that chitin only provided partial protection to *bak1-5* and no protection to *bkk1-1/bak1-5* plants is of interest since, to date, the LysM containing chitin receptor CERK1 is not known to use BAK1 or BKK1 for signalling (Liu *et al.* , 2012; Yasuda, Okada and Saijo, 2017). These data suggest that additional downstream signals linked to BAK1 are required for CERK1 signalling.

Both the chloroplast and light have an impact on plant resistance. Exposure of plants to high light causes rapid changes in nuclear gene expression in a photosynthesis-dependent manner and is associated with chloroplast-to-nucleus retrograde signalling (Suzuki *et al.* , 2012; Vogel *et al.* , 2014; Exposito-Rodriguez *et al.* , 2017). A 1 h high light treatment of *Nicotiana benthamiana* reduced F_v/F_m from 0.7 to 0.5 (Exposito-Rodriguez *et al.* , 2017), a more significant drop than we see with *A. thaliana* over a 3.5 h period (Figure 6C), most likely consistent with the higher light intensity of 1000 µmol $m^{-2}s^{-1}$ compared to 650 µmol $m^{-2}s^{-1}$ used in this study. Notably, this drop in F_v/F_m was accompanied by a 50% increase in H_2O_2 (Exposito-Rodriguez *et al.* , 2017). A genetically encoded H_2O_2 reporter localised to the stroma and nucleus revealed that high light treatment induced H_2O_2 production in these organelles for up to 1 h, and critically, the increase in nuclear H_2O_2 was dependant on electrons from the chloroplast (Exposito-Rodriguez *et al.* , 2017). High light conditions also induced perinuclear clustering of 7-8 chloroplast per nucleus, a similar observation as has been reported for plant-virus interactions (Caplan *et al.* , 2015; Ding *et al.* , 2019). It is predicted that this physical localisation facilitates the rapid diffusion of H_2O_2 from chloroplast to nucleus which elicits an alteration in nuclear gene expression (Exposito-Rodriguez *et al.* , 2017). Here we examined the effect of high light on *P. syringae* infection with the addition of peptide pre-treatments and ABA signalling. Our results show that high light has a synergistic effect with effector mediated suppression of F_v/F_m . Critically, MAMP pre-treatment or ABA co-infiltration fail to attenuate this suppression during DC3000 infection (Figure 6A-F, 7F, G and 9). F_v/F_m levels during a DC3000 *hrpA* infection also reduced significantly during the first 6 h of high light but recovered to 0.7, compared to 0.75 under normal light (Figure 6A-D). Furthermore, the co-receptor double mutant, *bkk1-1/bak1-5* , also showed increased F_v/F_m suppression compared to wild type following DC3000 challenge which was accentuated under high light (Figure 6 G-I). Strikingly, contrary to

expectations given the elevated H_2O_2 production, Col-0,*aao3* and *bkk1-1/bak1-5* lines all showed a significant increase in bacterial growth under high light whereas the hypersensitive*abi1/abi2/hab1* mutant and flg22 insensitive mutant *fls2*showed no increase in susceptibility to DC3000 infection compared to Col-0 under high light (Figure 6I, 7C). How and why these lines are insensitive to high light conditions warrants further investigation.

Complex plant hormone synthesis and signalling crosstalk play an important role in the outcome of plant disease and defence responses. Both salicylic acid (SA) and jasmonic acid (JA) are considered key hormones involved in plant immunity however, it has become apparent in recent years that ABA has a significant role to play in hormone manipulation during pathogen infection (Robert-Seilaniantz, Grant and Jones, 2011). Many organisms produce ABA, from cyanobacteria and fungi to humans, with kingdom specific synthesis pathways. In plants ABA is synthesised from carotenoids within the chloroplast, with the final two enzymatic reactions in the cytosol (Schwartz, Qin and Zeevaart, 2003; Finkelstein, 2013). As part of its virulence strategy, *P. syringae* induces *de novo* ABA biosynthesis *in planta* and this acts in part by suppressing SA biosynthesis and SA-mediated defences to aid disease progression (de Torres-Zabala *et al.* , 2007; De Torres Zabala *et al.* , 2009; Salomon *et al.* , 2014). Application of exogenous ABA (or coronatine) also induces the expression of the genes encoding three protein phosphatases 2C (PP2Cs), HAI1, HAI2, and HAI3 all of which interact with and inactivate MPK3 and MPK6, resulting in ABA-mediated MPK3/MPK6 immune suppression (Mine*et al.* , 2017). The PP2C triple mutant, *abi1/abi2/hab1* is ABA hypersensitive and has enhanced susceptibility to DC3000 whereas the ABA biosynthetic mutant *aao3* shows enhanced disease resistance (de Torres-Zabala *et al.* , 2007; De Torres Zabala *et al.* , 2009). Chlorophyll fluorescence allows dissection of the dynamics of these mutants during DC3000 infection, with the triple mutant exhibiting a stronger suppression of F_v/F_m (and a faster increase in NPQ) while the converse is true for the*aao3* mutant compared to Col-0 (Figure 7A, B, Supp Figure 3) (de Torres Zabala *et al.* , 2015). Notably, endogenous and exogenous ABA differentially impact apoplastic ROS production, with flg22 challenge of transgenic lines overexpressing ABA resulting in increased apoplastic H_2O_2 production, whereas plants with reduced ABA levels produced less apoplastic H_2O_2 following flg22 treatment (Tan*et al.* , 2019). By contrast, ABA pre-treatment resulted in a reduction in flg22 induced apoplastic H_2O_2 indicating that endogenous and exogenous ABA function differently during MAMP-induced apoplastic ROS burst in *A. thaliana* (Tan *et al.* , 2019). During a DC3000*hrpA* infection, cROS is produced 3-4hpi, whereas DC3000 infection suppresses cROS, but not when primed with elf18 or flg22 (Figure 3A, B) (de Torres Zabala *et al.* , 2015). Unexpectedly, cROS generation appeared ABA dose dependent, as leaves co-infiltrated with DC3000 and 10 µM ABA elicited a faster decrease ofF_v/F_m (similar to the hypersensitive *abi1/abi2/hab1* mutants).

Collectively, these data show thatF_v/F_m is a reliable, quantitative, real-time indication of pathogen infection and that abiotic factors affecting chloroplast functions e.g., high light and ABA (induced during drought and other abiotic stresses) generally result in reduced tolerance to bacterial infection.

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Conflict of Interest

The authors declare no conflict of interest.

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Author contributions

MG, SB, EB and RH conceptualised and designed the experiments. SB, EB, RH, SA and IA performed the

experiments. TG and HB generated material and provided experimental insights. SB, MG, EB prepared the manuscript.

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Figure Legends

Figure 1: MAMP pre-treatment protects F_v/F_m from bacterial induced suppression. A. Graph quantifying changes in F_v/F_m over 23 hpi with DC3000 infection on Col-0 leaves. Blue line represents leaves pre-treated with H_2O; orange - pre-treated with chitin (100 µg/ml); dashed grey - leaves pre-treated with flg22 (1 µM) and dashed yellow - leaves pre-treated with elf18 (1 µM). **B.** 18 hpi image F_v/F_m of a Col-0 plant pre-treated with H_2O, chitin (100 µg/ml), flg22 (1 µM) and elf18 (1 µM) at 18 hpi with DC3000. Orange represents normal F_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .**C.** Graph quantifying changes in F_v/F_m over 24 h following DC3000 infection on Col-0 and *efr1* leaves. Blue line represents Col-0 leaves pre-treated with H_2O; grey - Col-0 leaves pre-treated with flg22 (1 µM); yellow - Col-0 leaves pre-treated with elf18 (1 µM); green - *efr1* leaves pre-treated with H_2O; dashed blue - *efr1* leaves pre-treated with flg22 (1 µM) and red - *efr1* leaves pre-treated with elf18 (1 µM).**D.** Visual snapshot of F_v/F_m for a Col-0 plant (top right) and *efr1* plants pre-treated with H_2O, flg22 (1 µM) and elf18 (1 µM) at 18 hpi with DC3000. Orange represents normal F_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m . **E.** Graph quantifying changes in F_v/F_m over 24 h of DC3000 infection on Col-0 and *cerk1-2* leaves. Blue represent Col-0 leaves pre-treated with H_2O; orange - Col-0 leaves pre-treated with chitin (100 µg/ml); grey - Col-0 leaves pre-treated with flg22 (1 µM); green - *cerk1-2* leaves pre-treated with H_2O, red - *cerk1-2* leaves pre-treated with chitin (100 µg/ml) and dashed blue - *cerk1-2* leaves pre-treated with flg22 (1 µM). **F.** 18 hpi image of F_v/F_m for Col-0 (top right) and *cerk1-2* plants pre-treated with H_2O, flg22 (1 µM) and chitin (100 µg/ml) challenged with DC3000. Orange represents normal F_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m . Flg22; flg: chitin; chi: elf18; elf.

Figure 2: MAMP pre-treatment protects F_v/F_m from bacterial induced suppression, with the exception of chitin pre-treatment of *fls2* leaves. A. Graph quantifying changes in F_v/F_m over 24 hpi with DC3000 on Col-0 and *cerk1-2* leaves. Blue line represents Col-0 leaves pre-treated with H_2O; yellow - Col-0 leaves pre-treated with elf18 (1 µM); green - *cerk1-2* leaves pre-treated with H_2O and dashed red line - *cerk1-2* leaves pre-treated with elf18 (1 µM) **B.** Representative visual snapshot of F_v/F_m for a Col-0 plant (top right) and *cerk1-2* plants pre-treated with H_2O and elf18 (1 µM) at 18 hpi with DC3000. Orange represents normal F_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .**C.** Graph quantifying changes in F_v/F_m over 24 h of DC3000 infection on Col-0 and *efr1* leaves. Blue represents Col-0 leaves pre-treated with H_2O; orange - Col-0 leaves pre-treated with chitin (100 µg/ml); green - *efr1* leaves pre-treated with H_2O and red - *efr1* leaves pre-treated with chitin (100 µg/ml) **D.** Visual snapshot of F_v/F_m for a Col-0 plant (top right) and *efr1* plants pre-treated with H_2O and chitin (100 µg/ml) at 18 hpi with DC3000. Orange represents normal F_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .**E.** Graph quantifying changes in F_v/F_m over 24 h of DC3000 infection on Col-0 and *fls2* leaves. Blue represent Col-0 leaves pre-treated with H_2O; orange - Col-0 leaves pre-treated with chitin (100 µg/ml); grey - Col-0 leaves pre-treated with flg22 (1 µM); green - *fls2* leaves pre-treated with H_2O; dark blue - *fls2* leaves pre-treated with chitin (100 µg/ml) and red - *fls2* leaves pre-treated with flg22 (1 µM) **F.** Image of F_v/F_m for a Col-0 plant (top right) and *fls2* plants pre-treated with H_2O, chitin (100 µg/ml) and flg22 (1 µM) at 18 hpi with DC3000. Orange represents normal F_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .

Figure 3: DC3000 is unable to suppress cROS in MAMP pre-treatment leaves . Col-0 leaves treated with the non-specific species stain H_2DCF-DA 5.5 hpi with DC3000*hrpA* and DC3000. Leaves imaged on a Zeiss 880 using excitation at 488 nm, emission window of 512–527 nm to capture the oxidised dichlorofluorescein signal (Green). Chloroplast fluorescence was measured at 659–679 nm (red). Scale bars showing 20 μm. **A.**H_2O pre-treated and DC3000*hrpA* infection**B.** H_2O pre-treated and DC3000 infection**C.** flg22 (1 μM) pre-treated and DC3000 infection **D.**elf18 (1 μM) pre-treated and DC3000 infection. Image representative of three biological replicates.

Figure 4: Bacterial MAMP pre-treatments provide full and partial protection on *bkk1-1* and*bak1-5* single mutant lines but fail to protect*bak1-5/bkk1-1* lines. **A.** Graph quantifying changes inF_v/F_m over 23 h of DC3000 infection on Col-0 and *bkk1-1* leaves. Blue line represents Col-0 leaves pre-treated with H_2O; grey - Col-0 leaves pre-treated with flg22 (1 μM); yellow - Col-0 leaves pre-treated with elf18 (1 μM); green dashed line - *bkk1-1* leaves pre-treated with H_2O; dark blue - *bkk1-1* leaves pre-treated with flg22 (1 μM); red - *bkk1-1* leaves pre-treated with elf18 (1 μM)**B.** Image, 18 hpi with DC3000, ofF_v/F_m for Col-0 (top right) and*bkk1-1* plants pre-treated with H_2O and flg22 (1 μM). **C.** Image, 18 hpi with DC3000, ofF_v/F_m for Col-0 (top right) and*bkk1-1* plants pre-treated with H_2O and elf18 (1 μM). Orange represents normalF_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .**D.** Graph quantifying changes inF_v/F_m over 23 hpi with DC3000 on Col-0 and *bak1-5* leaves. Blue represents Col-0 leaves pre-treated with H_2O; grey - Col-0 leaves pre-treated with flg22 (1 μM); yellow - Col-0 leaves pre-treated with elf18 (1 μM); green -*bak1-5* leaves pre-treated with H_2O; dark blue -*bak1-5* leaves pre-treated with flg22 (1 μM) and red corresponds to *bak1-5* leaves pre-treated with elf18 (1 μM) **E.** Image at 18 hpi with DC3000, of $F_v/F_m$for Col-0 (top right), *bak1-5* (top left) or *bak1-5/bkk1-1*(bottom), plants pre-treated with H_2O and flg22 (1 μM).**F.** Image, 18 hpi with DC3000, ofF_v/F_m for Col-0 (top right),*bak1-5* (bottom) or *bak1-5/bkk1-1* (top left), plants pre-treated with H_2O and elf18 (1 μM) at 18 hpi with DC3000. Orange represents normalF_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .**G.** Quantitative changes inF_v/F_m over 23 hpi with DC3000 on Col-0 and *bak1-5/bkk1-1* leaves. Blue represents Col-0 leaves pre-treated with H_2O; grey - Col-0 leaves pre-treated with flg22 (1 μM); yellow - Col-0 leaves pre-treated with elf18 (1 μM); green - *bak1-5/bkk1-1* leaves pre-treated with H_2O; dark blue - *bak1-5/bkk1-1* leaves pre-treated with flg22 (1 μM) and red represents *bak1-5/bkk1-1* leaves pre-treated with elf18 (1 μM).

Figure 5: DAMP pre-treatment restricts suppression ofF_v/F_m following bacterial challenge. **A.** Graph quantifying changes inF_v/F_m over 24 hpi following DC3000 infection of Col-0. Blue represents leaves pre-treated with H_2O; orange - pre-treated with Pep1 (1 μM); dashed grey - pre-treated with Pep2 (1 μM) and yellow represents pre-treated with Pep3 (1 μM). **B.** Image ofF_v/F_m in Col-0 plants pre-treated with H_2O, Pep1 (1 μM), Pep2 (1 μM) or Pep3 (1 μM) at 18 hpi with DC3000. Orange represents normalF_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .**C.** Graph quantifying changes inF_v/F_m over 24 hpi with DC3000 on Col-0 and *pepR1-1x1-2* leaves. Blue represents Col-0 leaves pre-treated with H_2O; orange - Col-0 pre-treated with pep1 (1 μM); yellow - Col-0 pre-treated with pep3 (1 μM); red -*pepR1-1x1-2* pre-treated with H_2O; dark blue -*pepR1-1x1-2* pre-treated with pep1 (1 μM) and green represents*pepR1-1x1-2* leaves pre-treated with pep3 (1 μM). **D.**Image of F_v/F_m for Col-0 (right) and *pepR1-1x1-2* pre-treated with H_2O, Pep1 (1 μM), and Pep3 (1 μM) 18 hpi with DC3000. Orange represents normalF_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .**E.** Graph quantifying changes inF_v/F_m over 24 hpi with DC3000 on Col-0 and *pepR1-1x1-2* leaves. Blue represents Col-0 leaves pre-treated with H_2O; grey - Col-0 leaves pre-treated with flg22 (1 μM); red - *pepR1-1x1-2* leaves pre-treated with H_2O and dark blue - *pepR1-1x1-2* leaves pre-treated with flg22 (1 μM). **F.** Representative image, 18 hpi with DC3000, of F_v/F_m for Col-0 (right) and *pepR1-1x1-2* plants pre-treated with H_2O and flg22 (1 μM). Orange represents normalF_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .

Figure 6: High light enhances bacterial suppression ofF_v/F_m. **A.** Graph quantifying changes in F_v/F_m over 24 hpi of DC3000 (blue line) and DC3000*hrpA* (orange line) infection on Col-0 leaves under normal light (NL) (120 μmol $m^{-2}s^{-1}$). **B.** Image ofF_v/F_m at 23 hpi of Col-0 plants with DC3000 and DC3000*hrpA* under NL (120 μmol $m^{-2}s^{-1}$). Orange represents the expected F_v/F_m , whereas yellow/green/blue represents suppressedF_v/F_m . **C.** Graph quantifying changes in F_v/F_m over 26.5 hpi of DC3000 (blue line) and

DC3000*hrpA* (orange line) infection on Col-0 leaves under high light (HL) (650 µmol $m^{-2}s^{-1}$). **D.** Image ofF_v/F_m at 22.75 hpi of Col-0 plants with DC3000 and DC3000*hrpA* under HL (650 µmol $m^{-2}s^{-1}$). Unlabelled leaves are not infiltrated. Orange represents expectedF_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .**E.** Graph quantifying changes inF_v/F_m over 26.5 hpi of DC3000 on Col-0 under high light (HL) (650 µmol $m^{-2}s^{-1}$). Blue represents Col-0 leaves pre-treated with H_2O; grey - Col-0 leaves pre-treated with flg22 (1 µM) and the yellow line represents Col-0 leaves pre-treated with elf18 (1 µM). **F.** Image ofF_v/F_m for Col-0 plants pre-treated with H_2O, flg22 (1 µM) and elf18 (1 µM) at 22.75 hpi with DC3000 under HL (650 µmol $m^{-2}s^{-1}$). Orange represents expected F_v/F_m , whereas yellow/green/blue represents suppressedF_v/F_m . **G.** Graph quantifying changes in F_v/F_m over 26.5 hpi of Col-0 and *bak1-5/bkk1-1* leaves with DC3000 under high light (HL) (650 µmol $m^{-2}s^{-1}$). Blue represents Col-0 and red *bak1-5/bkk1-1* leaves **H.** Image of F_v/F_m 22.75 hpi with DC3000 on Col-0 plant (right) or *bak1-5/bkk1-1* plants under HL (650 µmol $m^{-2}s^{-1}$). Orange represents expected F_v/F_m , whereas yellow/green/blue represents suppressedF_v/F_m .

Figure 7: High light renders Col-0 and *bak1-5/bkk1-1* plants more susceptible to bacterial infection. Bacterial growth of DC3000 on Col-0, *fls2*and *bak1-5/bkk1-1* plants under normal light (NL; blue; 120 µmol $m^{-2}s^{-1}$) and high light (HL; red; 450 µmol $m^{-2}s^{-1}$). Error bars, mean ± SE (n=6), student t-test determined statistical significance of $P < 0.0001$ for NL Col-0 vs *fls2* and Col-0 vs*bak1-5/bkk1-1* (not shown on graph), HL Col-0 vs*bak1-5/bkk1-1,* Col-0 NL vs HL, *bak1-5/bkk1-1* NL vs HL. There was no significant difference between HL Col-0 vs *fls2* and*fls2* NL vs HL. Representative of three biological replicates.

Figure 8: Exogeneous ABA synergistically or antagonistically alters pathogen induced ABA suppression of F_v/F_m in a concentration dependent manner. A. Graph quantifying changes inF_v/F_m over 24 hpi following DC3000 or DC3000*hrpA* infection of Col-0, *aao3* and*abi1/abi2/hab1* leaves. Blue represents Col-0 leaves infiltrated with DC3000; red - Col-0 leaves infiltrated with DC3000*hrpA* ; grey - *aao3* leaves infiltrated with DC3000; yellow - *aao3*leaves infiltrated with DC3000*hrpA* ; dark blue -*abi1/abi2/hab1* leaves infiltrated with DC3000 and green corresponds to *abi1/abi2/hab1* leaves infiltrated with DC3000*hrpA* . **B.** Image ofF_v/F_m of Col-0, *aao3* and*abi1/abi2/hab1* plants 18 hpi with DC3000 or DC3000*hrpA* . Orange represents normal F_v/F_m readout, whereas yellow/green/blue represents suppressedF_v/F_m . **C.** Bacterial growth of DC3000 on Col-0, *aao3* or *abi1/abi2/hab1* plants under normal light (NL; 120 µmol $m^{-2}s^{-1}$; blue) or high light (HL; 450 µmol $m^{-2}s^{-1}$; orange) conditions. Error bars, mean ± SE (n=6) student t-test determined statistical significance of $P < 0.0001$ for Col-0 NL vs HL (shown), statistical significance of $P < 0.001$ for *aao3* NL vs HL and statistical significance of $P < 0.005$ for HL Col-0 vs*abi1/abi2/hab1* . Representative of three replicated experiments.**D.** Graph quantifying changes in F_v/F_m 24 hpi with DC3000 on Col-0 leaves in the presence of ABA under normal light (NL; 120 µmol $m^{-2}s^{-1}$). Blue - Col-0 leaves infiltrated with DC3000 and orange - Col-0 leaves co-infiltrated with DC3000 + 10 µM ABA. **E.** Image ofF_v/F_m of Col-0, plants 18 hpi infiltrated with DC3000 and co-infiltrated with DC3000 and 10 µM ABA under NL. Orange represents expectedF_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .**F.** Graph quantifying changes inF_v/F_m up to 24 hpi with DC3000 of Col-0 leaves in the presence of increasing concentrations of ABA under high light (HL; 650 µmol $m^{-2}s^{-1}$). Blue represents Col-0 leaves infiltrated with DC3000; orange - Col-0 leaves co-infiltrated with DC3000 + 10 µM ABA and grey corresponds to Col-0 leaves co-infiltrated with DC3000 + 100 µM ABA. **E.** Image, 19.5 hpi, of F_v/F_m of Col-0 infiltrated with DC3000, co-infiltrated with DC3000 and 10 or 100 µM ABA under HL. Orange represents expectedF_v/F_m , whereas yellow/green/blue represents suppressed F_v/F_m .

Figure 9: Schematic overview of findings from study. Black arrows show pathways to normalF_v/F_m while red arrows show pathways to suppressed F_v/F_m or increased bacterial growth. **A. MAMP pre-treatment followed by DC3000 infection on receptor mutant plants.** *fls2* leaves pre-treated with elf18 maintain normalF_v/F_m while fls2 leaves pre-treated with chitin, Pep1 or 2 show suppressedF_v/F_m . *efr1* leaves pre-treated with flg22 or chitin maintain normalF_v/F_m . *cerk1-2* leaves pre-treated with flg22 or elf18 maintain normalF_v/F_m . *PepR1-1x2-1* leaves with flg22 maintain normal F_v/F_m .**B. MAMP pre-treatment followed by DC3000 infection on MTI co-receptor mutant plants.** *bak1-5* leaves

pre-treated with flg22 or elf18 maintain normal F_v/F_mwhile *bak1-5* leaves pre-treated with chitin show suppressedF_v/F_m . *bkk1-1* leaves pre-treated with flg22, elf18 or chitin maintain normalF_v/F_m . *bak1-5/bkk1-1*leaves pre-treated with flg22, elf18, chitin, Pep1 or 2 show suppressedF_v/F_m . Under high light (HL) conditions (dashed red line) *bak1-5/bkk1-1* leaves show an increased suppression of F_v/F_m(thick, red dashed arrow) and increased bacterial growth compared to normal light (NL) conditions. **C. Chlorophyll fluorescence and bacterial growth are altered under different light conditions.** Under normal light (NL; 120 µmol $m^{-2}s^{-1}$) conditions DC3000*hrpA* infected leaves maintain normalF_v/F_m , while DC3000 infected leaves show suppressed F_v/F_m . Pre-treatment of Col-0 leaves with flg22 or elf18 under NL results in normal F_v/F_m . Under high light (HL; 650 µmol $m^{-2}s^{-1}$) DC3000*hrpA* , DC3000 and flg22 or elf18 pre-treated DC3000 infected leaves all show suppressed $F_v/F_m$and DC3000 infected leaves show an increase in bacterial growth.**D. Chlorophyll fluorescence is reduced during high light and ABA treatment.** Under normal light (NL; 120 µmol $m^{-2}s^{-1}$) conditions DC3000 infected leaves and leaves co-infiltrated with DC3000 + 10 µM show suppressedF_v/F_m . Under high light (HL; 650 µmol $m^{-2}s^{-1}$) leaves infected with DC3000, leaves co-infiltrated with DC3000 + 10 µM and leaves co-infiltrated with DC3000 + 100 µM all showed suppressedF_v/F_m . Created with BioRender.com.