The effect of abiotic stress on plant resistance to insect herbivores

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Layman's summary

Plants are stationary organisms meaning they cannot move away from hostile environments. Because of this, plants have evolved broad scale adaptations in order to improve their survivability when conditions change for the worse. However, climate change is threatening plant life as both the severity and frequency of hostile climate events increase. In addition, plants need to defend against insect herbivore attacks. Insect feeding accounts for an annual loss of up to twenty percent of total plant biomass. For this reason, to enhance insect resistance plants can increase their physical barriers and produce toxic compounds in response to insect herbivores. The plants ability to adapt to both hostile environments and to defend against insect attack complicates responses as signaling networks collide. The combined stress of changing environmental conditions and insect feeding lead to unpredictable adaptation as both influence one another. Crucial in this matter are phytohormone signaling and hormone crosstalk. The phytohormone jasmonic acid (JA) regulates transcriptional activation towards defense against both chewing and piercing insects. Additionally, JA signaling is involved in increasing plant tolerance towards heat stress, drought and UV-B radiation. During defense signaling, the JA response is co-regulated by either ethylene (ET) or abscisic acid (ABA). Furthermore, the co-regulation with ABA finetunes and steers the immune response towards insect herbivores whereas ET can antagonize it. Furthermore, the phytohormone salicylic acid (SA) is also involved in defense signaling towards biotrophic pathogens and piercing insects. In addition, both SA signaling as well as JA signaling can negatively regulate each other. The resulting hormone crosstalk allows for an extensive signaling network which regulates plant responses during the combined stress. In this manner, both heat stress and drought result in a synergistic increase in plant defenses towards insect herbivores. Individually, both heat stress and drought cause a surge in ABA, JA and SA levels involved in enhancing stress tolerance. The resulting phytohormone signaling indirectly leads to the JA-dependent production of anti-herbivore compounds. Moreover, when plants are subject to insect feeding as well as drought or heat stress, the defense response is enhanced even further. Besides drought and heat stress, UV-B radiation also leads to enhanced resistance towards insects. However, the potential enhanced defense response due to UV-B is plant-insect dependent. In contrast, both submergence and plant shading leads to increased susceptibility towards insects. Due to restricted gas diffusion during submergence, ET accumulates negatively influencing JA signaling. As a consequence the JA-mediated defense response against insect herbivores is inhibited. Similarly, shading causes JA levels to decrease, inhibiting plant defense responses. Because of this, insect herbivores grow better on plants subject to submergence or shading. A better understanding on how plant defenses towards insects are differentially regulated during hostile environmental conditions is thus crucial in this changing climate.

Abstract

Plants in the field are often subject to a combination of abiotic and biotic stress. During the combinatory stress plant phytohormone signaling cross-communicates in order to optimize development. The resulting hormone crosstalk leads to adaptation divergent from the individual stresses. For this reason, abiotic stress can influence plant resistance towards insect herbivores. During insect herbivore feeding, plant defense responses are initiated following recognition of D/HAMPs by PRRs. Jasmonic acid (JA) together with abscisic acid (ABA) form the core signaling pathway in plant resistance towards insect herbivores. Elevated JA and ABA levels lead to increased expression of *MYC*, *MYB*, *WRKY* transcription factors resulting in accumulation of defensive compounds. In addition, salicylic acid (SA) is involved in the defense against piercing insects and insect egg deposition. However, elevated SA levels antagonize JA signaling leading to increased insect susceptibility. Furthermore, elevated ethylene (ET) levels inhibit the expression of *MYC2* leading to increased susceptibility to insects. Both heat stress and drought result in enhanced JA and ABA biosynthesis and signaling. For this reason, both abiotic stresses synergistically increase glucosinolate and proteinase inhibitors accumulation resulting in increased defense against insect herbivores. In addition, UV-B radiating can also increase the accumulation of defensive compounds in a JA-dependent manner. In contrast, submergence leads to the suppression of JA biosynthesis. Moreover, SA-mediated *WRK22* expression during flooding further suppresses JA signaling. In addition, the accumulation of ET during submergence inhibits *MYC2* expression hampering defensive responses. Similarly, shading leads to a reduction of active JA levels due to sulfation. The reduced JA levels are found to result in a decreased accumulation of defensive compounds. In this manner, both submergence and shading increase plant susceptibility towards insect herbivores.

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Introduction

Due to their sessile lifestyle, plants cannot run away from the biotic and abiotic stresses of their environment. As a consequence plants have evolved sophisticated mechanisms to perceive their environment and adapt accordingly. For instance, when submerged during flooding events, plants elongate their internodes and form aerenchyma to keep their leaves above the water surface and increase diffusion in the roots (Loreti et al., 2016). In addition, during drought spells plant retain their water balance by closing their stomata and induce architectural changes in their root systems (Gupta et al., 2020). However, plant life is threatened by the constant increase in greenhouse gas emissions and subsequent increased global temperatures as they drive increased frequency and severity of abiotic stress (Coolen et al., 2016; Zandalinas et al., 2021). Among the most relevant are increases in heat stress, drought and submergence following flooding due to climate change. Besides abiotic stresses, plant survival is also threatened by insect herbivores owing to their abundance and diversity. Insect herbivores consume around twenty percent of the plant biomass produced annually (Agrawal, 2011). Furthermore, average crop losses from arthropod pests account for fifteen percent of annual production (Mitchell et al., 2016). Worse still, warmer temperatures cause increased winter survival of insects which allows them to build up populations more rapidly leading to increased insect outbreaks (Harvey et al., 2020).

The regulatory mechanisms leading to plant resilience to the abiotic and biotic stresses are extensively studied but field conditions are unlike conditions found in the laboratory (Thoen et al., 2017). In the field plants are not only subject to an individual stress but the combination of abiotic and biotic stresses. The resulting net impact of these combined stresses to plant health differs from individual stresses (Gupta et al., 2017). Furthermore, the elucidated regulatory networks to individual stresses cannot reliably predict transcriptional response to stress combinations (Thoen et al., 2017). Some responses are prioritized over others potentially causing antagonistic effects. For instance, it is known that the combination of drought and heat stress have markedly different physiological and molecular responses in plants than the individual stresses (Kissoudis et al., 2014; Suzuki et al., 2014). Additionally, acclimation to stress combinations are known to weaken plant defense against biotic infections (Suzuki et al., 2014). For example, the combination of drought spells with pathogen infections has been found to inflict a greater crop yield loss (Gupta et al., 2017). However, there are also reports of increased insect resistance during heat stress (Xie et al., 2020).

Central to the regulation and adaptation to biotic and abiotic stresses is the signaling mediated by phytohormones. Synergistic and antagonistic crosstalk between salicylic acid (SA) and jasmonic acid (JA) is key in switching the downstream plant defense response (Beyer et al., 2021; Cui et al., 2019; Erb & Reymond, 2019; Ye et al., 2021). Additional crosstalk between JA, ethylene (ET) and abscisic acid (ABA) further finetunes the immune response (Aerts et al., 2021; Proietti et al., 2018; Vos et al., 2015). Moreover, ABA and ET in addition to JA and SA are also involved in regulation of plant signaling during abiotic stress (Arif et al., 2020; Coolen et al., 2016; Rai et al., 2020; Thoen et al., 2017; J. Wang et al., 2020; Zandalinas et al., 2020). With the increasing significance of stress combinations, further understanding how abiotic stress influences plant insect resistance and the underlying role of hormone crosstalk is becoming more important than ever.

Overview plant immunity against insects

Plant insect perception and defensive response

Recognition followed by an efficient defense response against insect herbivores is essential for plant survival. In light of this, plants evolved pattern recognition receptors (PRRs) in order to perceive insects as well as microbes (Bigeard et al., 2015; Erb & Reymond, 2019; Ye et al., 2021). The receptors are able to recognize small conserved insect or pathogen derived molecules called pathogen-associated molecular patterns (PAMPS) to initiate early immune signaling events (Bigeard et al., 2015). However, since PRRs additionally recognize PAMPs from non-pathogens the term microbe-associated molecular patterns (MAMPs) has recently been adopted instead (Bigeard et al., 2015). In addition, plants perceive herbivores specifically through herbivore-associated molecular patterns (HAMPs) or by recognition of mechanical damage caused by chewing insects known as damage-associated molecular patterns (DAMPs) (Aerts et al., 2021; Erb & Reymond, 2019; Ye et al., 2021).

Localized at the cell-surface, PRRs trigger a process called pattern-triggered immunity (PTI) to initiate a common immune response against a broad spectrum insects and pathogens (Erb & Reymond, 2019; Ye et al., 2021). Three main groups of PRRs have been discovered, each able to recognize different molecular patterns; leucine-rich repeat-containing PRRs, binding molecules such as the bacterial flagellin; lysine motifs-containing PRRs, able to bind carbohydrate-based ligands such as insect chitin; and lectin-type PRRs binding to bacterial lipopolysaccharides (Ye et al., 2021). However, unlike PRRs known to signal for MAMPs, receptors involved in perception of HAMPs remain elusive. Notably the inceptin receptor (INR), a leucine-rich repeat receptor that confers signaling in response to common caterpillar oral secretions, has been discovered (Steinbrenner et al., 2020).

Next, in response to insect wounding and subsequent binding of D/HAMPs to PRRs, the PRRs are phosphorylated starting the PTI signaling. Starting with a burst of cytosolic Ca^{2+} resulting in the opening of other membrane channels. The ensuing influx and efflux of ions leads to depolarization of the plasma transmembrane (Bigeard et al., 2015; Erb & Reymond, 2019; Yan et al., 2018; Zebelo & Maffei, 2015). This is followed by induction of reactive oxygen species (ROS) and activation of mitogen-activated protein kinases (MAPKs) (Bigeard et al., 2015; Erb & Reymond, 2019; Zebelo & Maffei, 2015). The induction of both ROS and MAPKs activates important signaling pathways which lead to the production of anti-herbivore compounds and phytohormones involved in defense signaling (Bigeard et al., 2015; Escobar-Bravo et al., 2017; Zebelo & Maffei, 2015). In this manner, plant recognize and produce toxic secondary metabolites (glucosinolates and phenylpropanoids) in addition to proteinase inhibitors to defend against insect herbivores.

In addition to PTI, plants evolved a process called effector triggered immunity (ETI) to counteract insect or pathogen mediated suppression of PTI. During ETI, plants recognize insect salivary molecules called effectors which suppress PTI signaling setting off the immune response (Erb & Reymond, 2019). As well as ETI, plants additionally evolved an extensive signaling network to induce systemic regulation of defense responses following insect infestation. As insect

herbivores are mobile and often move to not attacked tissues, the systemic immune response effectively prepares plants against the insect predators (Erb & Reymond, 2019)

Role of JA in the core anti-herbivore defense signaling pathway

Phytohormones play an important role in downstream signaling and finetuning of immune responses by connecting perception and production of anti-herbivore compounds. The JA-mediated downstream response has been established as the core signaling pathway in defense against insect herbivores. JA signaling is involved in the positive regulation of resistance against leaf-chewing (caterpillars, larvae) and piercing (aphids, thrips, and mirid bugs) herbivore insects as well as phloem feeding arthropods (Aerts et al., 2021; Erb & Reymond, 2019; Escobar-Bravo et al., 2017; Howe et al., 2018; Ye et al., 2021). To enable the finetuning of the immune response, the JA pathway is divided in two branches which act with synergistic and antagonistic crosstalk. In reaction to necrotrophic pathogens, the branch co-regulated by ET signaling is activated (Aerts et al., 2021; Ye et al., 2021). Contrarily, when a plant is subject to insect herbivory the branch co-regulated by ABA is activated (Erb & Reymond, 2019). The JA/ABA branch signaling pathway is the main plant strategy against insect herbivores, whereas the JA/ET branch can inhibit plant insect defense signaling.

The molecular components underlying JA signaling have in part been elucidated (figure 1). Upon injury JA signaling is induced by the formation of the bioactive lipid jasmonoyl-L-isoleucine (JA-Ile) by JASMONOYL AMINO ACID CONJUGATE SYNTHASE (JAR1). Additionally, the biosynthesis of JA-Ile precursors is rate-limited by the regulation of both ALLENE OXIDE SYNTHASE (AOS) and LIPOXYGENASE (LOX) (Ruan et al., 2019). Next, JA-Ile is imported in the nucleus where it interacts with its coreceptor F-box protein CORONATINE INSENSITIVE 1 (COI1) and JASMONATE ZIM (JAZ) repressor (Erb & Reymond, 2019; Howe et al., 2018). In absence of JA-Ile, JAZ represses the JA downstream signaling pathway by competitively binding to the promoters of JA associated transcription factors (Erb & Reymond, 2019; Howe et al., 2018). In presence of JA-Ile, JAZ is degraded by COI1 in a E3 ubiquitin ligase SKP1-Cullin-F-box complex, lifting the repression of the MYC, MYB, ERF and WRKY transcription factor families (Ye et al., 2021). These transcription factors play a key role in regulation both up- and downstream of phytohormone signaling. The family of basic helix-loop-helix (bHLH) transcription factors MYC and MYB promote the expression of genes related to herbivory defense (Erb & Reymond, 2019; Ye et al., 2021). Co-regulated by ABA, MYCs act synergistically in the control of JA-dependent defenses (Aerts et al., 2021; Erb & Reymond, 2019; Wang et al., 2018). In their downstream signaling, MYCs promote the activity of MYB transcription factors (Erb & Reymond, 2019; Howe et al., 2018). In turn, MYBs act together with MYCs to regulate expression of the glucosinolate secondary metabolites essential for defense against insects (Erb & Reymond, 2019; Howe et al., 2018). Consistent with this, *myc* mutants show increased susceptivity to a large diversity of chewing insects (Erb & Reymond, 2019; Ye et al., 2021). For example, herbivorous insect caterpillar *Pieris rapae* preferred to feed from plants mutated in the MYC signaling branch (Vos et al., 2015). By contrast, ET stabilizes the ETHYLENE-INSENSITIVE 3 (EIN3) transcription factor which interacts and inhibits MYC genes (Song et al., 2014). This antagonistic interaction causes increased susceptibility to insects. Mutants in *ein3* fail to suppress the insect defense signaling pathway leading to increased resistance to the herbivore *Spodoptera exigua* (Song et al., 2014). The two JA co-regulated defense branches can be distinguished by regulation of the

marker genes *PLANT DEFENSIN 1.2* (*PDF1.2*) and *VEGETATIVE STORAGE PROTEIN 2* (*VSP2*). The MYC branch positively regulates the expression of MYC family of transcription factors and *VSP2*. While the presence of ET causes downregulation of VSP2 and MYC expression and instead upregulates downstream marker gene *PDF1.2* (Caarls et al., 2015). In this manner, the plant physiologically changes its defense strategy based on the specific pathogen.

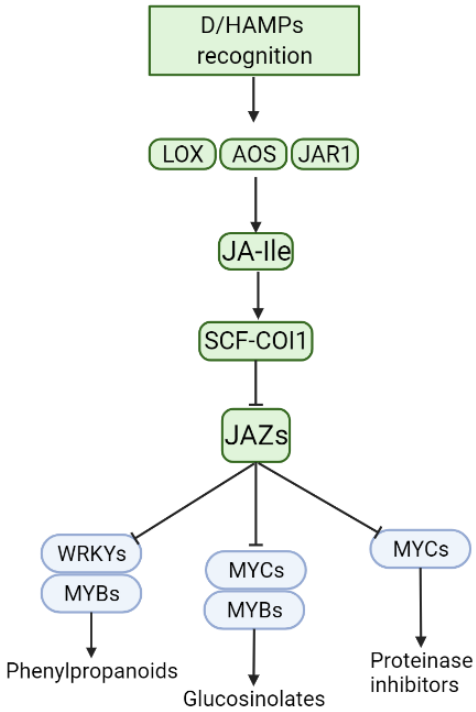

Figure 1. Model of JA signaling network in response to insect herbivores. *Recognition of D/HAMPS leads to the formation of active JA-Ile by the rate-limiting enzymes LOX, AOS and JAR1. Next, JA-Ile interacts with COI1 leading to degradation of the JAZ repressor. The subsequent derepressing of JA-mediated transcription factors leads to the production of anti-herbivore compounds (Erb & Reymond, 2019).*

Role of SA in insect herbivore defense signaling

Besides JA, SA is a key player in plant immune signaling in response to insect herbivores as well. the SA signaling pathway induces defense responses to fend off biotrophic and hemi-biotrophic pathogens as well as piercing insects (Aerts et al., 2021; Beyer et al., 2021; Cui et al., 2019; Erb & Reymond, 2019; Ye et al., 2021). In addition, SA is also reported to promote defense against mites (Resende et al., 2021). Intermediate concentrations of SA were found to enhance the physical barriers by increasing the thickness leaves and number of trichomes. However, the increased resistance to mites was lost when high doses of SA were applied (Resende et al., 2021). Besides its role in defense against mites, SA and ROS signaling were also found to be involved in the defense response to the deposition of insects eggs on leaves (Geuss et al., 2017; Gouhier-Darimont et al., 2019; Groux et al., 2021; Lortzing et al., 2019). The insect eggs are initially recognized by the PRRs LecRK-I.8 and close homolog LecRK-I1 (Gouhier-Darimont et al., 2019; Groux et al., 2021). Following recognition, the higher SA levels alter gene expression leading to the production of ovicidal substances which impair egg survival. Furthermore, SA signaling in response to oviposition can cause a hypersensitive response leading to cell necrosis (Fatouros et

al., 2014; Gouhier-Darimont et al., 2019; Groux et al., 2021; Lortzing et al., 2019). In addition, moderate levels of SA prime leaves that were previously egg-deposited. Despite the fact that defense signaling against chewing insects is mediated by JA, the defense priming from SA signaling results in enhanced defense against insect larvae (Lortzing et al., 2019). Consistent with this, mutants impaired in SA signaling are less able to defend against larvae after pre-deposition of eggs (Geuss et al., 2017; Gouhier-Darimont et al., 2019; Groux et al., 2021; Lortzing et al., 2019).

The SA-mediated transcriptional network has previously been elucidated (figure 2). SA is mainly synthesized from chorismic acid by ISOCHORISMATE SYNTHASE but also from the PHENYLALANINE AMMONIA LYASE pathway (Chen et al., 2009; Rai et al., 2020). Once high levels of SA are formed, it is transported to the cytosol by ENHANCED DISEASE SUSCEPTIBILITY 5 (EDS5) (Rai et al., 2020). Here it binds with its receptor and SA master regulator NONEXPRESSER OF PATHOGENESIS RELATED GENES1 (NPR1) (Arif et al., 2020; Caarls et al., 2015; Rai et al., 2020). NPR1 is present in the cytosol as an unactive oligomeric protein and binding of SA results in a breakup leading to monomeric NPR1. The activated monomeric NPR1 is transported into the nucleus where it act as regulator of the SA response (Arif et al., 2020; Rai et al., 2020). However, when SA levels are relatively low, NPR4 interacts and degrades the monomeric NPR1 proteins (Caarls et al., 2015). Just the same, when SA levels are too high, NPR3 binds with NPR1, also leading to degradation (Caarls et al., 2015). It is only at intermediate SA levels that NPR1 travels to the nucleus, activating the SA-mediated insect defense response (Caarls et al., 2015). Once in the nucleus, NPR1 binds with the leucine zipper form transcription factor TGA, which activates the transcription of downstream SA signaling genes (*PR1*, *PR5*, *EDS1*, *PAD4*, *WRKY70*) (Arif et al., 2020; Caarls et al., 2015; Rai et al., 2020). Subsequently, the transcription factor family WRKY acts in forming complex regulatory networks finetuning the SA-mediated transcriptional response (Arif et al., 2020; Hickman et al., 2019; Rai et al., 2020). Additionally, the thaumatin-like protein PATHOGENESIS-RELATED PROTEIN (PR5) is crucial in the defense against oviposition in response to egg deposition (Fatouros et al., 2014; Geuss et al., 2017; Lortzing et al., 2019). This is owing to the fact that PR5 regulates the biosynthesis of defensive metabolites (Geuss et al., 2017; Lortzing et al., 2019).

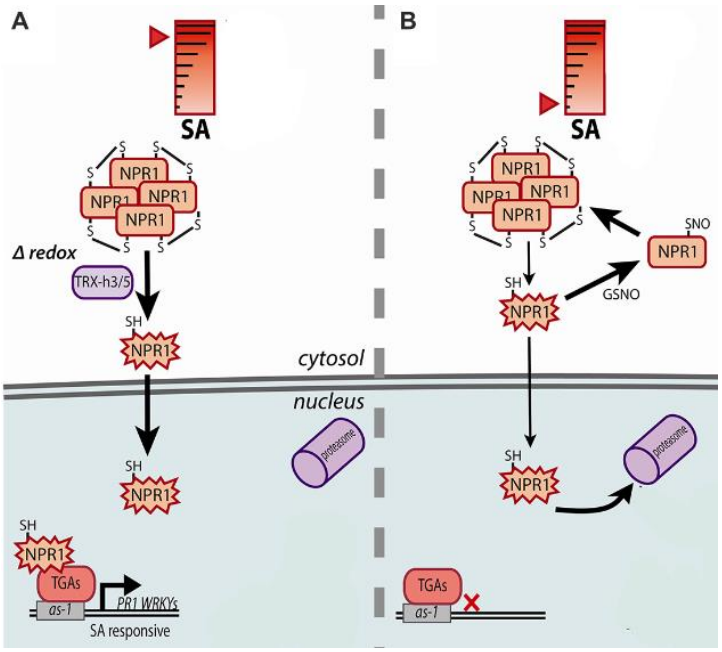

Figure 2. Model of SA signaling network. *Elevated levels of SA lead to the reduction and monomerization of NPR1. The activated NPR1 travels to the nucleus and together with TGAs induce the SA-mediated response (Caarls et al., 2015).*

Role of hormone crosstalk in response to abiotic and biotic stress

Phytohormone signaling is crucial as it allows plants to cross communicate different signaling pathways, each potentially acting synergistically or antagonistically from one another. The designated phytohormone crosstalk allows for enormous regulatory potential as it efficiently integrates and finetunes different signals to ensure optimal development. In this manner, hormonal crosstalk is thought to be evolved as a cost-saving strategy by suppressing unnecessary defense and developmental processes (Vos et al., 2015). In plant immunity the crosstalk between SA and JA, as well as between ET and ABA in steering the JA defense response, are classic examples (Figure 3). However, hormone crosstalk is broader than plant immune responses as the signaling in response to abiotic stress also influences the plant hormone homeostasis. For example, drought or elevated ABA levels antagonize the SA-mediated plant defense response subsequently leading to increased pathogen susceptibility (Gupta et al., 2017). Considering this, better understanding hormone crosstalk and the interactions between different signaling pathways is key in unraveling the impact of combined stress on plant insect immunity.

JA/SA crosstalk

Crosstalk between JA and SA modulates the most important switch in the plant immune system. However, both phytohormones also function in signaling outside of plant immunity. Besides plant defenses, SA also regulates a variety of physiological processes as it servers as an important

growth regulator under abiotic stress (Arif et al., 2020). In particular, by increasing proline, antioxidants and heat shock proteins, SA provides tolerance against temperature and drought related stresses (Arif et al., 2020). Owing to this fact, the SA-mediated *PR5* expression is also upregulated under drought stress enhancing drought tolerance (Lortzing et al., 2019). Similarly, JA signaling is also key in regulating different abiotic stresses (Kazan, 2015; J. Wang et al., 2020). Also important in drought tolerance, a JA precursor is involved in reducing stomatal aperture during drought, increasing plant survival (Savchenko et al., 2014). Furthermore, JA signaling has additionally been found to partly counteract the negative effects of UV-B stress (X. Liu et al., 2012).

In their crosstalk both SA and JA are able to influence each other's downstream signaling and hormone levels (Figure 3) (Beyer et al., 2021; Caarls et al., 2015; Hickman et al., 2019). However, SA signaling is dominant as it overrules JA signaling by inhibiting downstream JA signaling transcription factors (Beyer et al., 2021; Caarls et al., 2015; Hickman et al., 2019). In light of this fact an experiment was performed comparing the genes differentially expressed during individual and combined JA/SA application. The resulting JA responsive gene set was for 69% influenced by combined JA/SA application, while in contrast only 12% of the SA-mediated genes are differentially regulated by combined JA/SA application (Hickman et al., 2019). In addition, SA mediates suppression of JA signaling by altering DNA-binding abilities, degradation of proteins and by sequestering transcription factors away from the nucleus (Caarls et al., 2015).

To shed more light onto the molecular regulation of JA/SA crosstalk, multiple genes involved have been identified. The SA-mediated antagonistic effects on JA signaling is in part regulated through NPR1 and WRKY proteins. Downstream SA activity by WRKY51 inhibits the transcription of *AOS,* leading to suppression of JA biosynthesis and reduced defense against insects (Dong et al., 2003; Yan et al., 2018). In addition, SA also acts antagonistically on the JA/ET mediated defense response by negatively regulating the *ORA59* transcription factor (Zander et al., 2014). Following the suppression of *ORA59* by SA, plants show increased susceptibility to necrotrophic pathogens (Zander et al., 2014). In contrast, application of JA is less antagonizing to SA signaling. Nonetheless, immediately after JA application, genes related to SA signaling are downregulated (Hickman et al., 2017). Furthermore, the MYC branch of JA signaling suppresses SA biosynthesis by inhibiting the rate-limiting enzyme *ICS1* (Zheng et al., 2012). However, the interplay between SA and JA is not always antagonistic. Both SA and JA are essential for activation of ETI (L. Liu et al., 2016). During ETI, SA enhances the binding of NPR3 and NPR4 to JAZ leading to the degradation of JAZ and subsequent increase in JA signaling (L. Liu et al., 2016)

ABA/ET crosstalk

Better known for its major role in abiotic stress tolerance, ABA signaling is essential in plant resilience towards drought, heat stress, salinity, cold stress, UV-B and heavy metal (Vishwakarma et al., 2017). In addition, in a growth immunity trade off, ABA also balances the defense responses towards insects (Figure 3). In synergistic crosstalk with JA, ABA positively regulates the defense against chewing insects via positive regulation of *MYC2* expression and protein levels (Vos et al., 2015; Zander et al., 2020). As well as MYC2, ABA also co-regulates transcription factors of the NAC and MYB families, also involved in insect defense responses (Verma et al., 2016). In addition, ABA is also able to further stimulate defense signaling by indirectly inducing JA signaling by increasing the biosynthesis of a JA precursor (K. Wang et al., 2018). Moreover, ABA also negatively regulates defense responses against both biotrophic and necrotrophic pathogens. This

is controlled through antagonistic crosstalk with both SA and ET through negative regulation of their biosynthesis and signaling (Kissoudis et al., 2014). When ABA biosynthesis is impaired, inhibition of plant defense towards necrotrophic pathogens is no longer present (Proietti et al., 2018). In their negative crosstalk, ABA has also been found to suppress SAR by suppressing SA biosynthesis (Kissoudis et al., 2014). Interestingly, it was found that SA enhances the production of ABA in tomatoes under stressful conditions (Arif et al., 2020).

Like ABA, ET signaling is also involved in a variety of abiotic stresses antagonistic and synergistic crosstalk (Figure 3) (Roychoudhury & Tripathi, 2019). Important in this process is the ability of ET to enhance the level of oxidative stress under stress conditions (Arif et al., 2020; Roychoudhury & Tripathi, 2019). In addition, high ET levels also inhibit plant resistance towards herbivores by stabilizing ethylene-insensitive 3 (EIN3), which suppresses the MYC gene family (Ye et al., 2021). Furthermore, it was found that ET and SA have an antagonistic relationship as SA inhibits ET biosynthesis under stressful conditions (Arif et al., 2020). ET biosynthesis is also restricted at high SA levels under heat and salinity stress (Arif et al., 2020).

JA signaling is also influenced by antagonist crosstalk with gibberellic acid (GA). Regulated through negative interaction between JAZ and DELLA, GA plays a central role in abiotic and biotic stress crosstalk. High GA levels result in the degradation of DELLA proteins leading to indirect repression of JA signaling (Erb & Reymond, 2019). However, both ABA and ET signaling promote DELLA stabilization indirectly promoting JA signaling (Kissoudis et al., 2014).

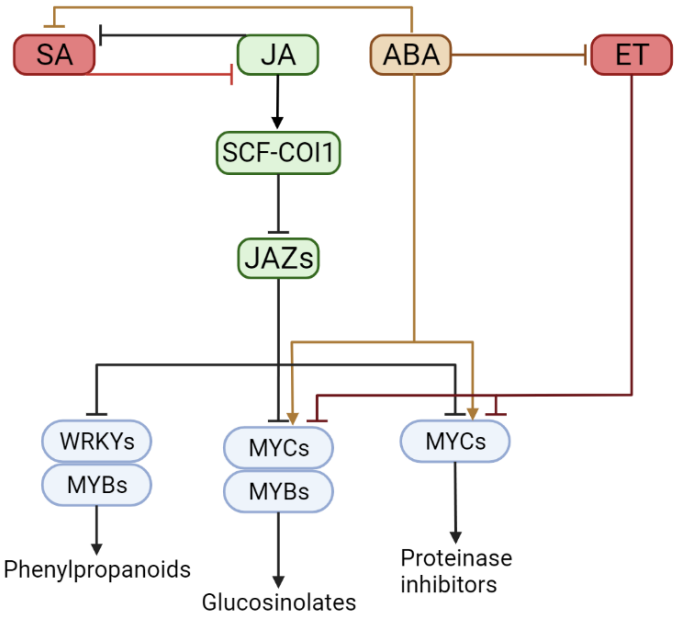

Figure 3. Simplified model of hormone crosstalk and plant signaling towards insect defense. *JA and SA negatively influence each others signaling and biosynthesis influencing plant defense responses. Additionally, ET antagonizes plant resistance towards insects. ABA positively regulates plant resistance towards insects whilst also antagonizing ET biosynthesis and signaling (Aerts et al., 2021; Arif et al., 2020; Erb & Reymond, 2019; Kissoudis et al., 2014).*

Effects of abiotic stresses on plant resistance against insect herbivores

Plants continuously adapt to their environment in order to ensure optimal development. Accordingly, plants require intelligent allocation of resources to deal with the different biotic and abiotic stress factors. Phytohormone signaling networks regulate both defensive capabilities and environmental resilience in a growth-defense tradeoff (Monson et al., 2022). Owing to this fact, phytohormone signaling plays a more dominant role in regulating transcriptomes during combined stresses than individual stresses (Coolen et al., 2016). In addition, plants react differently to combinatory stresses. Many components of the individual signaling networks function synergistically or antagonistically during combinatory stress. Furthermore, some stresses can be prioritized over others further complicating the plant signaling network (Coolen et al., 2016). Due to this complexity plant responses to combinatory stresses cannot be predicted from the individual stress (Thoen et al., 2017). However, plants do exhibit some common molecular responses during combinatory stresses reminiscent of the individual stress (Suzuki et al., 2014; Thoen et al., 2017). Most importantly, phytohormone signaling in response to abiotic stresses affects plant immune responses (Nguyen et al., 2016; Zhao et al., 2021). The resulting hormone crosstalk during different abiotic stresses and plant insect herbivory is thus key in better understanding plant responses to combinatory stresses.

The effect of heat stress on plant insect herbivore resistance

Physiological effects of heat stress

High temperatures are a serious threat to plants and crop production. The accelerated increase in temperatures due to global warming has enlarged this problem even further. The detrimental effect of high temperatures is termed heat stress (HS) and affects plant growth, development and yield (Nazar et al., 2017; Rai et al., 2020; Zhao et al., 2021). In particular, temperatures between 35-45°C lead to degradation of proteins and a decrease in protein synthesis as well as rapid production of ROS, eventually resulting in cell death (Nazar et al., 2017; Rai et al., 2020; Zhao et al., 2021). In addition, HS causes membrane fluidity leading to altered membrane-bound protein structures resulting in increased membrane permeability and severe cellular injury (Nazar et al., 2017; Rai et al., 2020). Furthermore, thylakoid membranes increase in fluidity hampering the electron transport and causing reduction in photosynthetic efficiency (Zhao et al., 2021). Moreover, the photosynthetic machinery also reduce in efficiency as a result of reduction in structure due to thermal instability (Zhao et al., 2021). Plants enhance HS tolerance by induction of osmolyte synthesis and increase in antioxidants needed to confront heat-induced oxidative damage (Haider et al., 2021; Nazar et al., 2017; Rai et al., 2020). The accumulation of the antioxidant proline is among the most important adaptive mechanisms in responses to HS. The increased proline levels enable higher water intake, lead to detoxification of excess ROS and protects the photosynthetic machinery during HS (Khan et al., 2013; Nazar et al., 2017).

Role of phytohormones in HS tolerance

Phytohormone signaling is crucial in mediating regulation of HS adaptation in plants. Both SA and ABA play a pivotal role in regulating increased plant resilience to HS (Arif et al., 2020; Haider et al., 2021; Nazar et al., 2017). When a plant is subjected to high temperatures, endogenous SA levels increase resulting in increased proline production (Khan et al., 2013; Nazar et al., 2017). In this manner, synthetic increase of SA levels have been found to cause increased heat tolerance in grape leaves and cucumber plants (Arif et al., 2020; Nazar et al., 2017). SA has also been found to restrict ET signaling by inhibiting biosynthesis under HS (Khan et al., 2013). Similar to SA, ABA signaling contributes to thermotolerance by minimizing damage to photosynthetic machinery and by increasing antioxidants (Jha et al., 2021). Besides SA and ABA, endogenous JA levels were also found to increase along with JA-mediated transcription during HS (Haider et al., 2021; Xie et al., 2020). As a result, the expression of SA- and JA-mediated *NAC*, *WRKY* and *MYB* transcription factors are induced during HS (Haider et al., 2021; Zhao et al., 2021). Together with the ABA regulated key *HEAT SHOCK TRANSCRIPTION FACTORs* (*HSFs*), these transcription factor families collectively form a complex transcriptional network to enhance HS tolerance in plants (Haider et al., 2021; Xie et al., 2020).

Asymmetric effect of HS on plant defense against insects

The effect of HS on plant defense and herbivore performance is asymmetric as the increased temperatures affect both insects and plants. The elevated temperatures result in increased growth rates and feeding needs of insects herbivores (Lemoine et al., 2014). The higher feeding needs are a result of reduced efficiency in converting plants into body mass at higher temperatures (Bauerfeind & Fischer, 2013; Lemoine et al., 2014). Furthermore, HS is found to have differing effects on insect herbivory rates depending on the plant-insect system used (Bauerfeind & Fischer, 2013; Lemoine et al., 2014; Paudel et al., 2020; Sun et al., 2018; Tian et al., 2021). Similarly, increased temperatures also affect plants as elevated temperature additively increases plant constitutive defense enzyme activities and glucosinolate levels (Paudel et al., 2020; Xie et al., 2020). As a result, HS is found to lead to relatively less insect growth and increased mortality despite the increased feeding habits. However, as HS results in increased insect feeding in addition to plant stress, cases of decreased plant biomass have been reported (Bauerfeind & Fischer, 2013; Lemoine et al., 2014; Paudel et al., 2020). Still, the reduction in insect growth during HS, together with the increase in defense signaling followed by biosynthesis of defensive compounds, does suggest HS leads to increased resistance to insect feeding (Paudel et al., 2020).

Role of phytohormones in defense signaling during HS

The increase in plant insect herbivore resistance is linked to an increase in JA and ABA levels (Xie et al., 2020). Specifically, the combination of HS and insect herbivory synergistically increases JA biosynthesis. The increase in JA biosynthesis is a result of upregulation in *AOS* and *LOX* expression (Xie et al., 2020). The increased JA levels regulate the rise in defensive compounds found during the combinatory stress and lead to increased resistance to aphid infestation (Xie et al., 2020). In this manner, HS leads to increased insect herbivore resistance due to a JA-mediated increase of anti-herbivore compounds found during HS.

The effect of drought on plant insect herbivore resistance

Physiological effects of drought

Water is crucial for plant survival and as such drought is arguably the most threatening abiotic stress for plants to overcome. Therefore plants have evolved multiple different strategies to enhance drought tolerance. Key in improving drought tolerance is increasing water uptake and maintaining the physiological water balance. As a result, drought spells induce large architecture changes in the root meristem, vasculature and stem cells (Gupta et al., 2020). Crucially, plants minimize water loss by closing the stomata in response to drought (Gupta et al., 2020). Plants additionally prevent cellular damage by producing peroxidase enzymes and proline metabolites to maintain membrane integrity and redox homeostasis (Gupta et al., 2020)

Role of phytohormones in drought tolerance

Similar to HS, the phytohormone ABA is key in regulating plant responses to drought (Gupta et al., 2020). Following dehydration, ABA biosynthesis is induced resulting in downstream signaling through SNF1-RELATED PROTEIN KINASE 2 (SNRK2) to increase water use efficiency (Gupta et al., 2020). In addition, SA is involved in the regulation of various growth parameters during drought stress (Arif et al., 2020; Gupta et al., 2017). Under drought stress, application of SA has been shown to enhance phenols and total soluble proteins in maize (Arif et al., 2020). Moreover, SA application has been shown to increase drought resistance by increasing relative water contents (Arif et al., 2020). Besides ABA and SA, JA signaling also is found to be involved in drought resilience (Jang et al., 2017). The biosynthesis and signaling of JA is found to be upregulated during drought (Gupta et al., 2017; Wang et al., 2021). As a result, JA-mediated *MYC2* expression is induced resulting in xylem differentiation (Jang et al., 2017). Moreover, both JA and ABA are involved in drought priming, a strategy enhancing drought tolerance (Wang et al., 2021). The accumulation of ABA and JA during drought also synergistically increase JA levels by inducing the expression of *AOS* and *LOX* (Xie et al., 2020). In addition, a precursor of JA functions together with ABA in promoting stomatal closure in response to drought (Savchenko et al., 2014).

Effect of drought on plant defense against insects

Following the combined drought and herbivore stress, the plant responds by inducing expression of stress-related processes and repressing growth (Coolen et al., 2016). This way, the plant prioritizes survivability over development in order to adapt to the changes in environment. It was found that the plant signaling against insect herbivory takes over transcriptome responses during drought in a rapid manner. Moreover, phytohormone signaling plays a dominant role in switching plant transcriptome responses. The resulting switch is found to lead to increased insect resistance (Coolen et al., 2016). Furthermore, drought is found to induce plant defense responses to insect herbivores irrespective of additional stresses (Gupta et al., 2017; Nguyen et al., 2016; Xie et al., 2020). As such, the production of defensive compounds such as protein inhibitors are increased following drought (Nguyen et al., 2016). Yet similar to HS, the increased insect resistance leads to a growth tradeoff (Bauerfeind & Fischer, 2013; Lin et al., 2021; Paudel et al., 2020). Plants subject to the combined stress have relatively lower compensatory regrowth than plants solely subject to herbivory (Coolen et al., 2016; Lin et al., 2021). Consistent with the growth-defense

tradeoff, plants under drought stress has been found to cause stunted growth and reduced plant biomass (Nguyen et al., 2016).

Role of phytohormones in defense signaling during drought

Plant phytohormone signaling in response to insect herbivores is mediated differently during drought. Also further divided between plant-insect models, drought in combination with herbivore whiteflies do not synergistically act to increase endogenous ABA levels in *Arabidopsis thaliana* (Gupta et al., 2017). Contrarily, both aphid infestation and *S. exigua* larvae were found to synergistically increase ABA biosynthesis in wheat and *Solanum dulcamara* (Nguyen et al., 2016; Xie et al., 2020). Nevertheless, feeding of all three plant-insect models resulted in an initial burst of JA after which it displayed similar constitutive high levels (Gupta et al., 2017; Xie et al., 2020; Nguyen et al., 2016). Similarly, the elevated SA levels and downstream signaling in response to drought are suppressed during herbivore infestation (Gupta et al., 2017; Nguyen et al., 2016). The resulting increase in JA levels and signaling combined with the decrease of SA levels and signaling results in the increased production of defensive compounds (Gupta et al., 2017; Nguyen et al., 2016; Xie et al., 2020). As well as increasing defensive compounds, drought additionally induces priming of insect defenses. Following drought, JA levels in addition to JA-defense responses are enhanced during insect infestation, indicative of a primed defense response (Coolen et al., 2016; Gupta et al., 2017; Xie et al., 2020). The resulting increased expression of defensive proteins leads to lower herbivore performance and increased mortality (Gupta et al., 2017; Lin et al., 2021; Nguyen et al., 2016; Xie et al., 2020). Altogether, the combined stress results in drought-mediated constitutive high ABA levels together with a burst of JA signaling and repression of SA biosynthesis. The subsequent signaling leads to increased insect defense and defense priming resulting in reduced insect growth and increased mortality (Gupta et al., 2017; Xie et al., 2020).

The effect of submergence on plant insect herbivore resistance

Physiological effects of submergence

Flooding and submergence are a disaster to plants and crops and lead to significant losses as plants cannot survive prolonged submergence. During submergence plants are covered in water resulting in oxygen deprivation. The lack of oxygen, called hypoxia, inhibits plant respiration resulting in reduced growth and eventually leading to death (Loreti et al., 2016). Moreover, the covered tissues also experience reduced light availability further inhibiting growth. During flooding events, plants adapt by either using a quiescence strategy or escape strategy. In the former plants keep their metabolism to a minimum until they re-emerge from the water. Contrarily, plants can display rapid vertical elongation to regain aerial contact and keep their leaves above the water surface. In addition, to adapt to the low oxygen environment plants can induce development of root aerenchyma in order to improve gas diffusion (Loreti et al., 2016).

Role of phytohormones in submergence tolerance

During submergence diffusion is restricted resulting in high accumulation of ET. Furthermore, ET biosynthesis is increased during submergence causing even further elevation in ET

concentrations (Loreti et al., 2016; Minami et al., 2018). The high ET levels downregulate antioxidant production leading to high ROS levels. The subsequent accumulation of ET and ROS modulate the signaling cascades during submergence (Loreti et al., 2016; Minami et al., 2018). In this manner, ET and ROS signaling leads to either the quiescence or escape strategy during submergence (Loreti et al., 2016). In addition, JA signaling is involved in growth suppression and acts as negative regulator of internode elongation during submergence (Minami et al., 2018). As a result, JA biosynthesis and signaling is decreased during submergence (Lee et al., 2020; Minami et al., 2018).

Effect of submergence on plant defense against insects

The combination of submergence followed by insect herbivores is found to have a negative effect on plant resistance (Hsu et al., 2013; Lee et al., 2020; Nguyen et al., 2016). Both larval weight and numbers increase ensuing flooding (Lee et al., 2020; Nguyen et al., 2016). Moreover, flooding suppresses plant insect defense responses. The negative regulation of JA following submergence is found to negatively impact plant resistance to herbivores (Lee et al., 2020; Nguyen et al., 2016). In addition, JA-mediated defense responses are further suppressed due to negative crosstalk between SA and JA. Submergence leads to the increased expression of members of the SA-mediated WRKY transcription factors in *A. thaliana.* (Hsu et al., 2013; Kloth et al., 2016). The ensuing upregulation of *WRK22* in turn results in increased defense responses against biotroph pathogens (Hsu et al., 2013). However, the induced *WRKY22* transcription factor is found to modulate interplay between JA-SA crosstalk (Kloth et al., 2016). Specifically, WRKY22 was found to act as a suppressor of JA signaling (Kloth et al., 2016). The resulting negative crosstalk leads to reduced resistance to aphid infestation (Kloth et al., 2016). In this manner, submergences results in increased resistance to biotrophic pathogens but antagonized herbivore defense responses (Hsu et al., 2013; Kloth et al., 2016). Moreover, the accumulation of ET during submergence is also found to add to the suppression of herbivore induced JA biosynthesis and signaling (Lee et al., 2020). The additional inactivation of JA-mediated herbivore defenses is regulated by ETHYLENE INSENSITIVE 2 (EIN2) in an ET dependent manner (Lee et al., 2020). Following the accumulation of EIN2, the anti-herbivore MYC branch is suppressed as MYC2 is degraded (Lee et al., 2020). Altogether, the combined suppression of JA signaling by ET and SA during flooding results in increased susceptibility to insect herbivores (Hsu et al., 2013; Kloth et al., 2016; Lee et al., 2020; Nguyen et al., 2016)

The effect of far-red light on plant insect herbivore resistance

FR-induced susceptibility

As photoautotrophic organisms, light is essential for plant growth and survival. For this reason light is a strong signal able to change plant development (Ballaré & Pierik, 2017). Plants sense light via photoreceptors called Phytochromes. After red (R) light is absorbed by chlorophyll, the phytochromes activate, starting a cascade of downstream transcriptional processes. In contrast to red light which is needed for photosynthesis, far-red (FR) light (700-780nm) is not absorbed causing plants to reflect it. The resulting low R:FR ratio is used by plants to detect neighboring plants and leads elongation in a response called shade avoidance (Ballaré & Pierik, 2017).

Additionally, the perception of far-red light and inactivation of photoreceptor phyB affects plant immunity (Chico et al., 2014; Fernández-Calvo et al., 2011; Fernández-Milmanda et al., 2020; Moreno et al., 2009). Termed FR-induced susceptibility, this growth-defense trade-off is associated with decreased resistance against herbivores. The resulting drop in herbivore resistance is problematic in agroecosystems where crop densities are high (Campos et al., 2016). For instance, plants grown under low R:FR ratios were shown to have increased susceptibility to *Spodoptera frugiperda* and *Spodoptera littoralis* caterpillar feeding (Fernández-Calvo et al., 2011; Moreno et al., 2009).

The role of phytohormones in FR induced susceptibility

The decrease in plant insect herbivore defense is in part due to a decrease in JA signaling (Fernández-Calvo et al., 2011; Fernández-Milmanda et al., 2020; Moreno et al., 2009). However, FR radiation is found to only affect the initial burst of JA signaling induced by wounding (Fernández-Milmanda et al., 2020). The lack of a fast JA-mediated defense response is the result of a reduction of jasmonate precursors due to sulfation by SULFOTRANSFERASE 2a (ST2a). Upregulated by low FR:R and subsequent inactivation of phyB, ST2a catalyzes the sulfation of JA leading to a reduction of active JA-Ile and inhibition of the JA defense response (Fernández-Milmanda et al., 2020). In addition, the JA signaling cascade is further repressed due to the degradation of DELLA proteins during FR enrichment (Chico et al., 2014; Fernández-Calvo et al., 2011; Fernández-Milmanda et al., 2020). Following the degradation of DELLA, JAZ proteins engage with the MYC2 key transcription factor, resulting in inhibition of the JA defensive response (Chico et al., 2014; Fernández-Calvo et al., 2011). In this manner, the low R:FR present in agroecosystems suppresses JA-mediated defense responses against insect herbivores resulting in increased susceptibility.

The effect of UV-B radiation on plant insect herbivore resistance

Physiological effects of UV-B

Ultraviolet-B (UV-B) light (280-315nm) is an important light signal in plants which influences the development of plants in a species specific manner. In lower doses, UV-B radiation induces photomorphogenic changes. However, at high intensities UV-B damages the plant and becomes a stress signal (Meyer et al., 2021). Among the changes in development at high intensities are suppression of hypocotyl elongation and induction of DNA repair. During UV-B stress growth is limited by inhibition of cellular division (Meyer et al., 2021; Vandenbussche et al., 2018) Moreover, plants respond to high UV-B light by inducing production of secondary metabolites to counteract and protect against oxidative damage by ROS (X. Liu et al., 2012; Meyer et al., 2021). In addition, to reduce UV-B penetration of the cell, plants accumulate UV-B photoprotective compounds and increase cell wall thickness (Escobar-Bravo et al., 2017; Meyer et al., 2021). The photomorphogenic and physiological responses are regulated by the UV-B specific photoreceptor UV RESISTANT LOCUS (UVR8) (Escobar-Bravo et al., 2017; Meyer et al., 2021). In response to UV-B radiation, the UVR8 photoreceptor converts to its active form and is translocated to the nucleus (Meyer et al., 2021). Once in the nucleus, UVR8 activates the downstream UV-B induced response.

Role of phytohormones during UV-B stress

Besides activation of UVR8, UV-B light has been found to induce the biosynthesis of both JA and SA (Meyer et al., 2021). However, the role of both phytohormones is not clear as contrasting results are reported. (Meyer et al., 2021). In *A. thaliana,* expression of multiple JA biosynthesis related genes are upregulated by UVR8 under UV-B light (Meyer et al., 2021). In addition, application of exogenous JA is found to increase plant tolerance to UV-B. Particularly, application of JA resulted in increased proline content leading to improved redox control during UV-B stress (X. Liu et al., 2012). The induction of SA during UV-B radiation is found to be species specific as not all accessions of *A. thaliana* show accumulation of SA in response to UV-B (Meyer et al., 2021). Moreover, only relatively low intensity UV-B radiation results in SA accumulation (Meyer et al., 2021).

Effect of UV-B on insect herbivore defense

The effect of UV-B light on plant herbivore defenses is mixed and plant-insect model dependent. The variation in reported results is in part due to differences in UV-B intensity, timing, duration and intensity of background light (Meyer et al., 2021). Nevertheless, UV-B light is demonstrated to positively influence plant resistance against sucking herbivores (Demkura et al., 2010; Đinh et al., 2013; Vandenbussche et al., 2018). Following UV-B radiation, plants induce production of phenolic compounds commonly described to increase plant resistance to herbivorous insects (Schreiner et al., 2012). In addition, UV-B-mediated photomorphogenic changes lead to improved plant defenses by inducing reinforced cell walls and biosynthesis of secondary metabolites (Vandenbussche et al., 2018). The resulting altered plant architecture and physiology positively impacts the plants resistance towards herbivorous arthropods (Vandenbussche et al., 2018). Contrarily, the effect of UV-B on chewing insects is less clear. In *A. thaliana* the increased flavonoid levels following UV-B radiation leads to reduced growth of *P. rapae* and *S. litura* larvae (Grant-Petersson & Renwick, 1996; Qi et al., 2018). However, feeding of *Trichoplusia ni* larvae was unaltered (Grant-Petersson & Renwick, 1996). Similarly, UV-B radiation did not result in increased resistance against *S. littoralis* larvae (Vandenbussche et al., 2018).

Role of phytohormones in defense signaling during UV-B stress

UV-B light combined with insect infestation can synergistically induce a burst of JA signaling. (Đinh et al., 2013; Schreiner et al., 2012; Vandenbussche et al., 2018). Moreover, following the burst of JA signaling, JA-dependent defensive compounds are found to accumulate (Đinh et al., 2013; Qi et al., 2018; Vandenbussche et al., 2018). In addition, UV-B light is able to prime JA-dependent insect herbivore defense responses (Demkura et al., 2010; Schreiner et al., 2012). Consequently, the defense against *S. litura* caterpillars is enhanced in a JA-dependent manner as a result of increased glucosinate contents due to UV-B radiation (Qi et al., 2018). Additionally, the priming of JA defenses positively influences resistance against mirid infestation (Đinh et al., 2013). Nevertheless, the resulting enhanced JA signaling does not increase resistance against *S. littoralis* (Vandenbussche et al., 2018). The lack of resistance is reasoned to be because of a lack of defensive compounds specific for *S. littoralis* (Vandenbussche et al., 2018). However, resistance against *M. persicae* aphids was enhanced due to UV-B. The increased resistance is hypothesized to be related to increased SA levels (Vandenbussche et al., 2018). A possible explanation for the

increased resistance could be the increased resistance to *M. persicae* oviposition due to the elevated SA levels (Fatouros et al., 2014; Geuss et al., 2017).

Discussion

Global warming is threatening plant life as the frequency and severity of abiotic stresses is increasing (Zandalinas et al., 2021). To adapt to the hostile environmental conditions plants have evolved multiple tolerance strategies in a growth-tolerance tradeoff (Monson et al., 2022). However, in addition to the increase in abiotic stress, plants are also threatened by biotic stresses. Insect herbivory accounts for twenty percent of annual plant biomass loss (Agrawal, 2011). Similar to the adaption to abiotic stresses, plants have evolved extensive immune responses to insect herbivory as well (Erb & Reymond, 2019). The induced immune response following insect infestation greatly enhances plant resistance to the insect herbivore threat. However, the increased frequency of abiotic stress is complicating plant immune responses as combinatory abiotic and biotic stress events increase. Moreover, plant signaling and responses during combinatory stress are different from the individual stresses (Coolen et al., 2016; Thoen et al., 2017). Regulatory mechanisms enhancing abiotic stress tolerance can work synergistically or antagonistically of defense signaling and vice versa. Therefore, a better understanding of the effect of abiotic stress on plant insect immune signaling is essential.

Plants recognize insects using cell surface bound PRRs. Following binding of H/DAMPS to the PRRs, early immune signaling cascades are initiated resulting in the production of anti-herbivore compounds and phytohormones (Erb & Reymond, 2019). The ensuing phytohormone signaling leads to systemic resistance and additional defensive responses. JA signaling forms the core signaling network involved in increased resistance against both chewing and sucking insect herbivores (Erb & Reymond, 2019). Additionally co-regulated by ABA, JA/ABA signaling induces expression of the *MYC* transcription factor family resulting increased production of glucosinolates and proteinase inhibitors (Aerts et al., 2021; Erb & Reymond, 2019). Besides ABA, JA defensive signaling is also finetuned by ET to steer plant defense towards necrotrophic pathogens. Furthermore, to effectively defend against biotrophic pathogens biosynthesis and signaling of SA is induced (Aerts et al., 2021). However, SA is also involved in defense against insects (Aerts et al., 2021; Erb & Reymond, 2019). SA can positively regulate defense against piercing insects and deposition of insect eggs (Erb & Reymond, 2019; Ueda et al., 2019).

Phytohormone crosstalk forms the backbone in the extensive signaling network finetuning plant growth and development (Aerts et al., 2021; Coolen et al., 2016; Vos et al., 2015). During plant immune responses both SA signaling and JA signaling work antagonistically from one another (Aerts et al., 2021; Erb & Reymond, 2019). As a result, upregulation of SA induces suppression of plant insect immune responses. Similarly, in co-regulating immune responses ET also suppresses JA signaling and MYC-regulated insect defenses (Erb & Reymond, 2019). In contrast, ABA co-regulates the expression of *MYC2* in insect defense responses (Zander et al., 2020). Besides immunity, SA also functions as growth regulator under abiotic stress. Together with stress phytohormone ABA, SA signaling plays a role during both HS and drought tolerance (Arif et al., 2020; Nazar et al., 2017). Similarly, JA signaling is also involved in drought, HS and UV-B tolerance (Gupta et al., 2017; Haider et al., 2021; Meyer et al., 2021). Because of this, plant resistance towards insect herbivores is affected by abiotic stress (Nguyen et al., 2016; Thoen et al., 2017; Zhao et al., 2021). Moreover, defense responses against insect herbivores are further

complicated by antagonistic or synergistic regulation of signaling due to combinatory abiotic and biotic stress (Coolen et al., 2016; Thoen et al., 2017).

The effect of abiotic stress on plant signaling against insect herbivores differs depending on the abiotic stress combination. During both HS and drought the biosynthesis and signaling of ABA, SA and JA increase (Haider et al., 2021; Nazar et al., 2017; Xie et al., 2020). The increased phytohormone levels enhance thermotolerance by increase proline production and reducing cellular damage (Haider et al., 2021; Xie et al., 2020). Similarly, during drought both water use efficiency and uptake are increased due to the phytohormone signaling (Gupta et al., 2017, 2020). Moreover, the enhanced JA/ABA levels and signaling during HS and drought additionally results in increased accumulation of anti-herbivore compounds (Gupta et al., 2017; Nguyen et al., 2016; Paudel et al., 2020; Xie et al., 2020). Furthermore, the defense response synergistically increases during the combined abiotic and biotic stress (Figure 4) (Coolen et al., 2016; Gupta et al., 2020; Xie et al., 2020). The combination results in further elevation of JA levels whilst SA levels decrease (Gupta et al., 2017; Xie et al., 2020). As a result, the combination of insect infestation and drought or HS leads to increased resistance towards both chewing insects and piercing insects (Coolen et al., 2016; Gupta et al., 2020; Xie et al., 2020). Both abiotic stresses resulted in decreased insect feeding whilst insect mortality increased (Bauerfeind & Fischer, 2013; Paudel et al., 2020; Xie et al., 2020). However, the increased resistance results in hampered plant growth due to a growth/defense tradeoff (Lin et al., 2021; Paudel et al., 2020). Similarly, UV-B stress is also found to increase plant resistance towards certain insect herbivores (Figure 4) (Demkura et al., 2010; Qi et al., 2018; Vandenbussche et al., 2018). Although the effect of UV-B on plants is dependent on methods used, UV-B is found to increase plant resistance towards piercing insects in addition to certain caterpillars. The increase in resistance is a result of a synergetic burst in JA signaling following the combined stress (Đinh et al., 2013; Schreiner et al., 2012; Vandenbussche et al., 2018). However, due to unknown reasons the resistance in plant-insect models are not all positively effected. This difference can potentially be explained due to differential effects of UV-B on plants (Meyer et al., 2021). Differences in UV-B intensities and supplemental light can have distinctly different effects on plants. As a result, both phytohormone biosynthesis and signaling can vary depending on models used (Meyer et al., 2021). The differential effect of UV-B on plant defenses against insects can thus potentially be explained by the varying regulation of JA-dependent defense signaling.

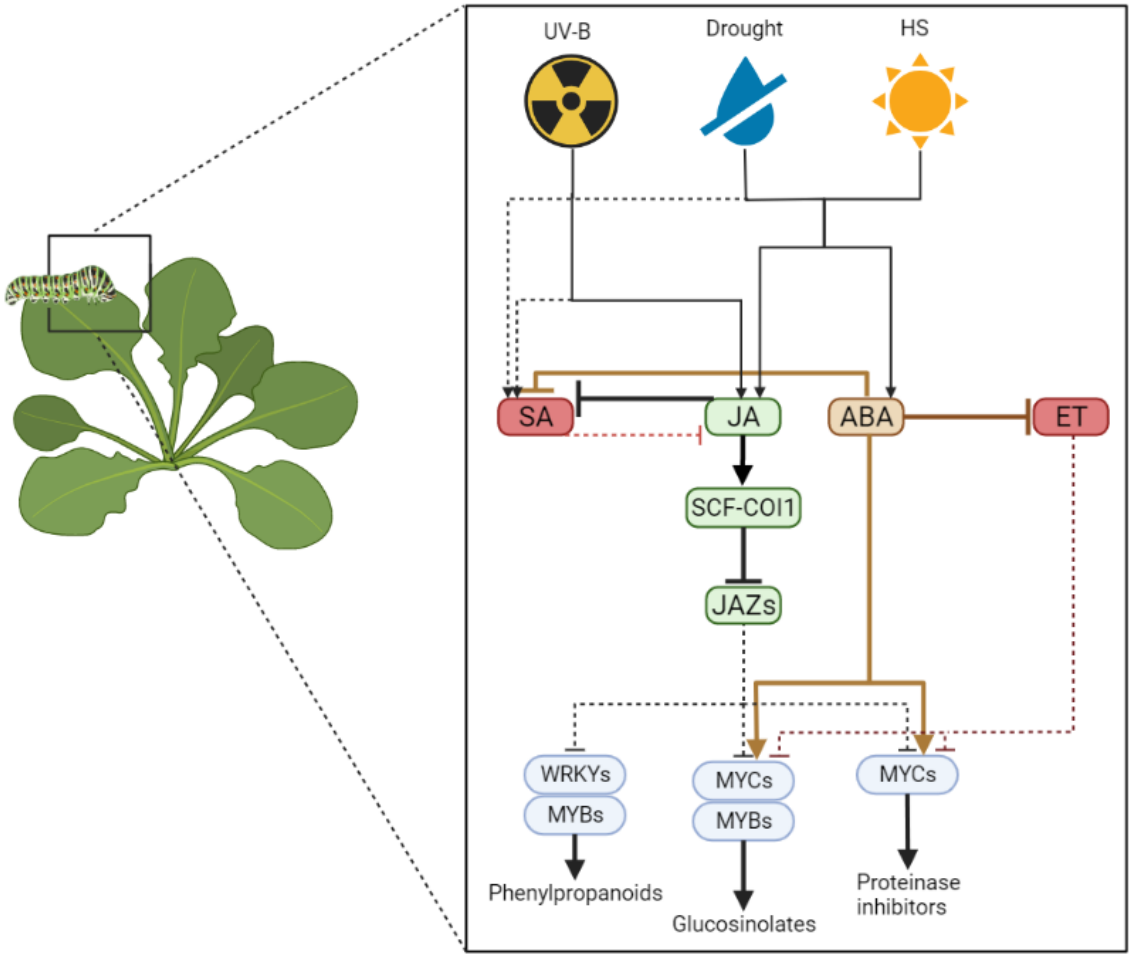

Figure 4. Simplified model of the effects of HS, drought or UV-B on plant defense responses against insect herbivores. *Following recognition of insect herbivores, JA biosynthesis and signaling is upregulated. Accumulation of JA leads to the degradation of JAZ proteins and subsequent depression of the JA-mediated downstream insect defense response. Enhanced ABA levels additionally upregulate the downstream response. The combination of drought or HS with insect herbivores leads to additional upregulation of JA and ABA biosynthesis and signaling. Unlike during the individual stress, SA biosynthesis is downregulated when combined with insect herbivory. The increased phytohormone levels lead to an enhanced defense response and accumulation of defensive compounds. Similarly, UV-B is also found to enhance plant resistance towards insects in a JA-dependent manner (Gupta et al., 2017; Vandenbussche et al., 2018; Xie et al., 2020).*

In contrast, both submergence and FR light lead to increased susceptibility towards insect herbivores (Figure 5) (Chico et al., 2014; Courbier et al., 2020; Lee et al., 2020). The high accumulation of ET during submergence results in antagonistic suppression of JA-mediated MYC2 activity (Lee et al., 2020). In addition, upregulation of SA-mediated *WRK22* suppresses JA signaling leading to increased insect herbivore susceptibility (Hsu et al., 2013; Kloth et al., 2016). Similarly, low R:FR ratios suppress JA signaling by reducing active JA levels. The low R:FR induces *ST2a* expression leading to sulfation and inactivation of JA precursors. The subsequent low JA levels inhibits the plants signaling leading to insect herbivore susceptibility (Chico et al., 2014; Fernández-Milmanda et al., 2020). In this manner, plants grown in agroecosystems have a problematic increased susceptibility towards insects as high plant densities result in shade

induced suppression of JA signaling (Campos et al., 2016). In addition, crop production in flood prone areas is further threatened as insect herbivore performance increases following submergence (Lee et al., 2020; Nguyen et al., 2016)

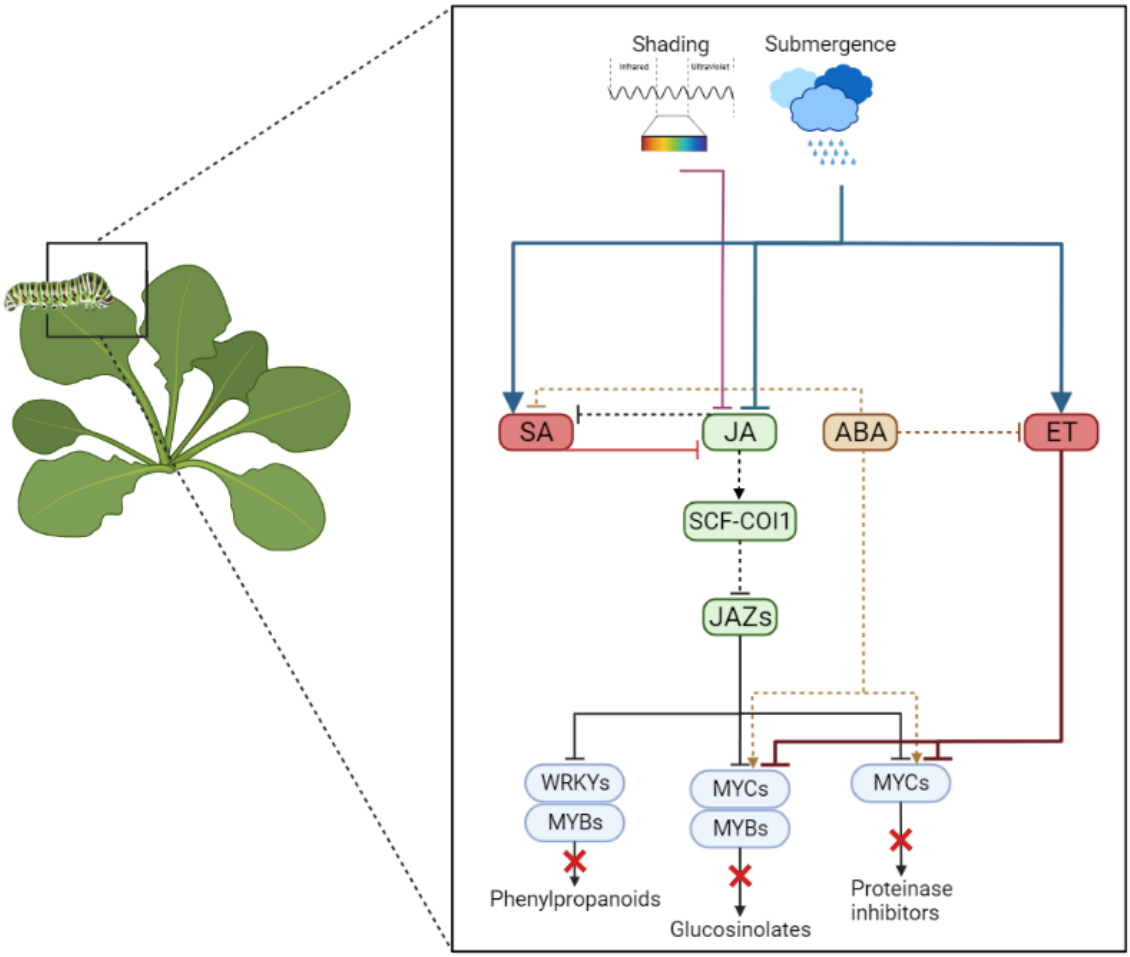

Figure 5. Simplified model of the effects of shading or submergence on plant defense responses against insect herbivores. *Following recognition of insect herbivores, JA biosynthesis and signaling is upregulated. Accumulation of JA leads to the degradation of JAZ proteins and subsequent depression of the JA-mediated downstream insect defense response. Enhanced ABA levels additionally upregulate the downstream response. Shading leads to a decrease in active JA levels repressing the JA-mediated degradation of JAZ proteins. Similarly, submergence inhibits the degradation of JAZ proteins due to lower JA levels. Additionally, the accumulation of SA-mediated WRKY22 during submergence further represses JA signaling. Moreover, the accumulation of ET leads to inhibition of MYC2 expression and subsequent production of defensive compounds. In this manner, both shading and submergence lead to increased insect herbivore susceptibility by inhibiting the defense response (Chico et al., 2014; Kloth et al., 2016; Lee et al., 2020).*

To gain more insight into the effects of stress combinations more research is needed on how different signaling networks cross communicate. Nearly all research is done on single stresses whilst plant stresses in the field are often not singular (Thoen et al., 2017). Furthermore, a better understanding on how different abiotic stresses influence plant immunity towards insect is essential. Often research on plant-insect systems during abiotic stress is restricted to the

accumulation of defensive compounds and insect survivability. Moreover, the inconsistent use of model plant-insect systems further complicates broad scale conclusions. In addition, knowledge on the regulation of phytohormone biosynthesis and signaling is still rudimentary. Research on phytohormones besides JA during stress combinations is even more elusive. As hormone crosstalk forms the backbone in regulation of combinatory stress, knowledge on the biosynthesis and signaling of ABA, SA and ET is essential. Elucidating the potential hormone crosstalk should lead to a better understand of the regulatory mechanisms during combinatory stress. Moreover, insights on regulation of downstream targets is also lacking. The regulation of *MYCs, NACs, WRKYs and VSP2* during combinatory stress is mostly unknown. Altogether, further exploration of the mechanisms behind regulation of stress combinations could lead to improved plant resistance towards insect herbivores whilst minimizing developmental costs.

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