Review

Jasmonate-induced defenses: a tale of intelligence, collaborators and rascals

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Plants have sophisticated defense systems to protect their tissues against the attack of herbivorous organisms. Many of these defenses are orchestrated by the oxylipin jasmonate. A growing body of evidence indicates that the expression of jasmonate-induced responses is tightly regulated by the ecological context of the plant. Ecological information is provided by molecular signals that indicate the nature of the attacker, the value of the attacked organs, phytochrome status and thereby proximity of competing plants, association with beneficial organisms and history of plant interactions with pathogens and herbivores. This review discusses recent advances in this field and highlights the need to map the activities of informational modulators to specific control points within our emerging model of jasmonate signaling.

Jasmonate (JA) signaling

JA is an oxylipin involved in the regulation of several physiological processes [1]. JAs were first connected with defense responses by the work of E.E. Farmer and C.A. Ryan [2,3] on the regulation of the expression of digestive proteinase inhibitors in the tomato (*Solanum lycopersicum*). Two decades after that exciting discovery, it is now firmly established that JA is a key cellular signal involved in the activation of immune responses to most insect herbivores and necrotrophic microorganisms (see Box 1 for defense-related terminology). The early signaling steps involved in the perception of the attack by herbivores or pathogens, through the detection of herbivore- and damage-associated molecular patterns (HAMPs and DAMPs, respectively) [4], and the activation of JA biosynthesis remain to be elucidated [5]. However, the details of the mechanism used by plant cells to perceive elevated levels of bioactive JA and transduce the JA signal into the activation of transcriptional responses have been thoroughly investigated in the past few years. Recent work has also demonstrated that JA signaling is finely controlled by internal and external signals that provide the plant with information about its physiological status and ecological context. Therefore, an emerging major challenge is to find the functional links between these ecological regulators and the key molecular players involved in the control of JA responses. Whereas recent reviews have covered particular aspects of the regulation of JA responses, including cross-talk with other hormones [6–8] and modulation by light signals [9], we still lack an integrated picture of plausible interactions among ecological regulators and the connections between these regulators and the recently discovered molecular components of the JA response.

In this review, I will focus on this active area of research and attempt to generate a preliminary map of the signaling circuits that modulate JA-induced defenses as a function of physiological signals and plant interactions with a diverse array of ecological actors, including consumers, competitors and beneficial organisms.

JA perception

The core events of JA perception (Figure 1) have recently been identified [10–16] and are discussed in several review papers [1,17–19]. Briefly, the perception of jasmonoyl-isoleucine (JA-Ile), the bioactive amino acid conjugate of jasmonic acid, is achieved by the ubiquitin ligase SCF^{COI1} complex. When the F-box protein CORONATINE-INSENSITIVE1 (COI1) recognizes JA-Ile, it triggers the ubiquitination and subsequent proteosomal degradation of JASMONATE ZIM DOMAIN (JAZ) proteins. The degradation of JAZ proteins relieves JAZ-mediated repression of gene expression, leading to the activation of JA responses (Figure 1). Recent structural and pharmacological studies [16] have indicated that the complex of both COI1 and JAZ (Figure 1) should be considered the true JA-Ile receptor. This is because COI1 contains an open pocket that recognizes JA-Ile, but a loop region in the JAZ protein is necessary to trap the hormone in this binding pocket; furthermore, these studies identified a third crucial component of the JA coreceptor complex, inositol pentakisphosphate, which interacts with both COI1 and JAZ adjacent to the ligand [16]. The activation of JA-responsive genes leads to the production of metabolites involved in defense. In addition, the local activation of the JA signaling cascade produces signaling molecules that propagate systemically to induce JA responses in organs not directly affected by the initial event of herbivory or pathogen infection, and provide protection against future attacks [5,20].

Adaptive modulation

One of the key aspects of the plant defense response is that its expression is modulated by the ecological context of the plant. Thus, the timing, intensity and characteristics of the defense repertoire are influenced by the specific nature of the attacker, the vitality of the attacked organs, the proximity of competing plants, the association with beneficial organisms and the history of previous interactions with

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Box 1. Basic glossary for plant enemies and defenses

Plant enemies

Plants are targeted by a wide spectrum of consumer organisms, which include pathogens (such as viruses, bacteria, fungi and oomycetes) and pests (such as herbivorous insects and nematodes). Plant pathogens are generally divided into *biotrophs* and *necrotrophs* according to their lifestyles [108]. Biotrophs derive their nutrients from living plant tissue, frequently by using haustoria that penetrate the plant cells without causing extensive cell damage. Necrotrophs first kill the host cells, often through the production of specialized toxins, and then feed on the remains. Some plant pathogens can display both lifestyles (e.g. depending on the stage of their life cycle), and are termed *hemibiotrophs*. Herbivorous insects are frequently divided according to their feeding styles into *biting–chewing* insects (which bite off and chew their food) or *sucking* insects (which pierce the plant tissue using specialized mouth parts and absorb liquid cell contents) [109].

Constitutive and induced defenses

Plants have several lines of *constitutive* (i.e. preformed) structural and chemical defenses to prevent attacks from a diverse array of enemies. If these initial layers of defense are broken, plants rely on a complex and highly regulated system of *inducible* responses (i.e. responses that are activated only when the plant is under attack by pathogens or herbivores). The expression of these induced defenses is coordinated by defense-related hormones.

Defense-related hormones (JA, SA and ET)

In addition to JA, which coordinates the defense responses against chewing insects (often termed the 'wound' response) and necrotrophic pathogens, two other hormones play key roles in orchestrating the expression of the plant immune system: SA and ET [7,108]. The SA response pathway is activated by plants in response to attack by pathogens with biotrophic lifestyles [108]. The plant defense response is initiated upon the recognition of pathogen signatures (PAMPS and microbial effector proteins) and often involves an increase in the levels of SA, programmed cell death at the site of infection and the accumulation of antimicrobial metabolites in systemic tissues (including PR proteins). The accumulation of defenses in tissues distal from the site of infection is called systemic-acquired resistance (SAR) and this confers broad-spectrum, long-lasting resistance against microbial pathogens. ET is a gaseous plant hormone whose production is frequently induced in response to attack by necrotrophic pathogens and some types of herbivore insects, as well by several stressors and environmental and developmental signals. ET plays an important role as a modulator of SA and JA responses [7,25,43].

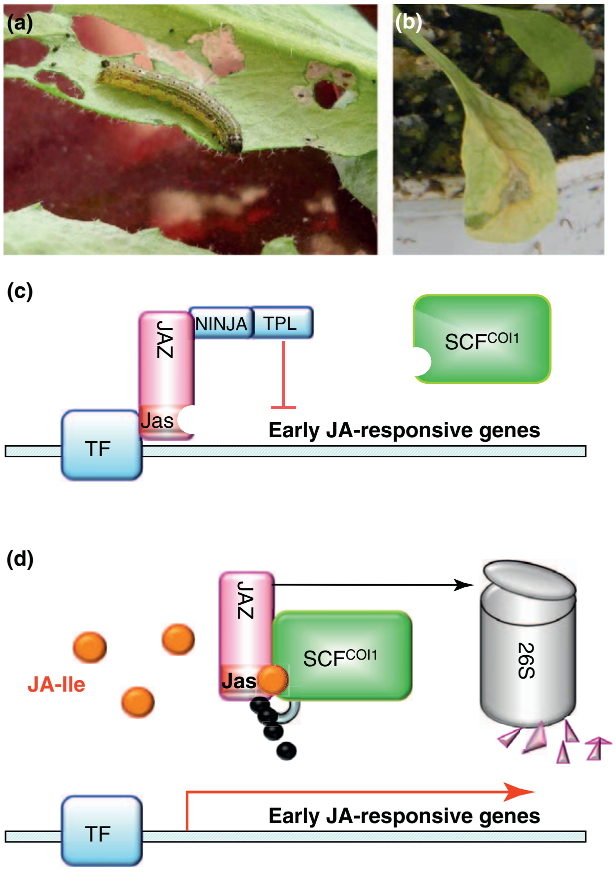

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Figure 1. The activation of the JA pathway by chewing insects and necrotrophic pathogens and the basic mechanism of JA perception. **(a)** *Arabidopsis* plants attacked by *Spodoptera frugiperda*, a chewing insect and **(b)** *Botrytis cinerea*, a necrotrophic fungus. **(c,d)** The activation of the JA pathway by the targeted degradation of JAZ repressor proteins. In the absence of bioactive JA (c), the transcription of defense-related genes is repressed by the interaction of the relevant transcription factors with JAZ proteins, which control gene expression through their interaction with the NINJA adaptor and TPL corepressor proteins [15], and presumably other repression mechanisms [19]. In the presence of the bioactive hormone (JA-Ile, orange circles) (d), JAZ proteins interact via their Jas domains with the SCF^{COI1} ubiquitin ligase, which targets them for proteasomal degradation, derepressing the transcription of defense genes (adapted from [15]). Recent structural and pharmacological evidence [16] supports the view that the true JA-Ile receptor is the COI1–JAZ complex, along with the cofactor inositol pentakisphosphate (not shown in the figure).

pathogens and herbivores. This regulation of plant defense is a clear example of the role played by information-acquiring systems in tailoring adaptive plant behavior [9,21,22].

Recent work has suggested that the plastic regulation of plant defense expression is achieved, at least in part, through the intricate, multilayer modulation of the JA signaling pathway. Our understanding of this modulation by environmental and internal signals, which is outlined in Figure 2, is still limited. In the following sections, I will discuss some of the key features and regulators of JA responses that contribute to the adaptive adjustment of the defense strategy of the plant.

Intelligent focusing: choosing the defense strategy

The activation of plant defenses implies allocation and ecological costs [23–27]. For example, the allocation of resources to defense against one type of attacker can reduce the ability of the plant to respond to the challenge of a different invader. Plants seem to use mechanisms that effectively adjust their defense repertoires on the basis of the characteristics of their attackers. These mechanisms of 'intelligent focusing' are frequently mediated by hormonal crosstalk [7,8,25,28–30].

A thoroughly characterized case of signal crosstalk is the antagonistic interaction between the JA and salicylic acid (SA) signaling pathways [7,25,28]. Plants infected by SA-inducing biotrophic pathogens often suppress JA-dependent defenses [31,32], apparently prioritizing the investment of resources in SA-dependent defense over JA-dependent responses (Figure 2). Similarly, the elicitation of the JA pathway can repress the SA response [33,34].

The mechanisms whereby SA modulates the JA response have been the subject of intense investigations and are discussed in several recent reviews [7,8,29]. Briefly, SA can depress both JA biosynthesis and sensitivity [35]. The downregulation of JA biosynthesis is thought to be a byproduct of the reduced JA sensitivity, because several JA biosynthetic genes are positively regulated by

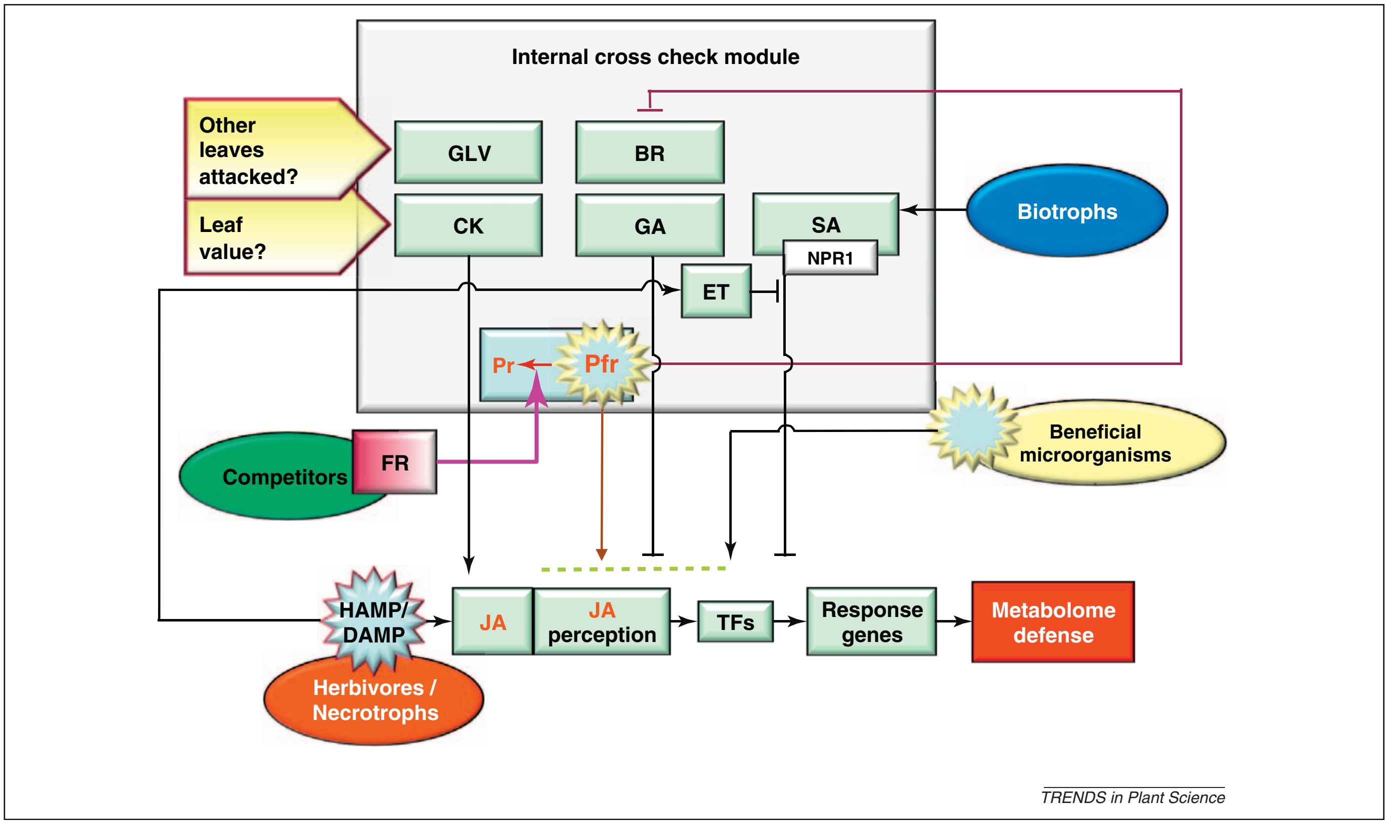

Figure 2. The modulation of the JA response by internal and external signals. The perception of HAMPs and DAMPs when plants are under attack by herbivores and necrotrophs activates the JA biosynthetic pathway. The production of bioactive JA-Ile leads to the derepression of the relevant transcription factors and defense-related genes, and the activation of plant defenses. The expression of the JA response is modulated by several physiological and ecological signals. The proximity of competing plants increases the levels of FR radiation, which weakens JA responses by converting the active, Pfr form of phyB (a positive regulator of JA sensitivity) into the inactive Pr form. Attacks from pathogens with a biotrophic lifestyle activate the SA pathway, which generally antagonizes the JA response. The SA effect is modulated by ET, which is another hormone frequently induced by the plant in response to HAMPs and DAMPs. Plant association with beneficial microorganisms can increase the JA response, presumably by increasing the abundance of defense-related transcription factors. The levels of several additional hormones contain information on the internal status of the plant tissue to be defended, and also regulate JA responses. BR and GA (two growth-promoting hormones) repress JA responses; this interaction might play a role in regulating the growth vs. defense allocation tradeoff. CK is a positive regulator of the JA response. CK concentrations are typically low in shaded and old leaves, and along with Pfr levels CKs might help concentrate the defense response in the most photosynthetically active (valuable) leaf elements. Defense responses are also potentiated by volatile signals (such as GLVs) produced by herbivore-attacked neighboring leaves. Arrows indicate promotion/activation; truncated lines indicate repression or negative interactions. For some regulators, the precise point of interaction with the JA signaling pathway is unknown, which is indicated by the dotted green line at the site of convergence. BR, brassinosteroid; CK, cytokinin; ET, ethylene; GA, gibberellin; GLV, green leaf volatiles; SA, salicylate; TF, transcription factor.

JA, and it does not seem to be required for the SA-mediated suppression of JA signaling [36]. The protein NPR1 (for NONEXPRESSOR OF PATHOGENESIS-RELATED GENES1) plays an important role in mediating the suppressive effects of SA downstream of JA. NPR1 is a crucial component of SA signaling, which is activated by SA-induced redox changes that reduce inactive NPR1 oligomers to active monomers. NPR1 monomers are translocated to the nucleus, where they function as coactivators of TGA transcription factors that regulate the expression of SA-responsive genes, including *PR* genes [37]. The role of NPR1 in the repression of JA responses has been investigated by elegant experiments carried out in *Arabidopsis* (*Arabidopsis thaliana*), which were inspired by the structural analogies between NPR1 and IκB [35]. IκB is a protein that plays a key role in regulating the effects of SA and aspirin as suppressors of the formation of prostaglandins, which are animal oxylipins involved in inflammatory responses to infection and tissue damage. Those experiments demonstrated that NPR1 is required for the suppressive effects of SA on pathogen-induced JA accumulation and JA-induced defense gene expression. However, nuclear localization was not required for the suppression of JA signaling, indicating that the effects of SA on JA signaling are mediated through the activity of NPR1 in the cytosol [35,38].

The molecular mechanisms that mediate the effects of NPR1 suppressing JA-dependent defenses remain to be identified [7,30]. Pharmacological and mutant studies in *Arabidopsis* have suggested that the NPR1-dependent effect of SA on JA signaling occurs downstream of the initial events of JA perception by the SCF^{COI1} complex and JAZ degradation (Figure 1), and probably targets the GCC box in JA-responsive genes [39]. The GCC box is a binding site for the transcription factors of the APETALA2/ETHYLENE RESPONSE FACTOR (AP2/ERF) superfamily that regulate the JA-induced response of several genes involved in plant defense, such as the plant defensin *PDF1.2*. NPR1-independent negative effects of SA on JA signaling have been reported [39,40], and recent studies have also shown that ethylene (ET) can bypass the requirement for NPR1 in the SA-induced depression of JA response [41]. In *Nicotiana attenuata* plants exposed to herbivore attack, NPR1 downregulates SA production; this has been interpreted as a strategy evolved by the plant to prevent the depression of JA-induced defenses by herbivores that activate the SA pathway (see 'The rascals') [42].

ET is another important modulator of JA-induced defense responses [7,25,43] (Figure 2). ET and JA frequently show a synergistic interaction in the induction of certain plant defenses, such as the upregulation of *PDF1.2* in *Arabidopsis* [44]. The synergistic effect between JA and ET has been attributed to the concomitant activation of common transcription factors, such as those that belong to the AP2/ERF superfamily in *Arabidopsis* [45,46] and the HD-Zip HAHB4 in the sunflower (*Helianthus annuus*) [47]. However, cases of negative interactions (ET inhibiting JA-induced responses) have also been reported [48]. In addition, ET can cancel the effect of SA as a downregulator of JA responses. For example, a recent study has shown that the activation of the ET pathway renders the plant insensitive to the suppressive effects of SA on JA-induced defenses [49]. The mechanism is not clear, but this ET effect (canceling the dampening effect of SA) might ensure that the defense response mounted by the plant against necrotrophic pathogens and certain herbivorous insects (which simultaneously induce both the JA and ET signaling pathways [48,50]) is not suppressed by subsequent attack or secondary infections by biotrophic pathogens.

Competition: balancing risks

In many ecological scenarios, competition with neighbors is a major determinant of the fitness of each individual plant. The allocation of photoassimilates and other resources to competition can limit the investment in defense, thereby increasing vulnerability to herbivores; similarly, allocation to defense can reduce competitive ability against neighboring plants [51–54]. This allocation compromise is often referred to as the ‘dilemma of plants’ [9,23,55].

Plants detect the proximity of competitors using photoreceptors, which are sensitive to changes in specific wavelengths in the canopy light environment produced by the proximity of chlorophyll-containing tissues. In particular, the absorption of red light (R) by chlorophyll and the scattering of far-red radiation (FR) by leaf tissues leads to a reduction in the R:FR ratio, signaling the proximity of plant foliage and thereby the potential competition for light. Plants can read this spectral signal using phytochromes, particularly phytochrome B (phyB). Low R:FR ratios inactivate phyB by depleting the levels of the active (Pfr) form of the photoreceptor, which leads to the expression of the shade-avoidance syndrome (SAS) [9,56–58]. The SAS is characterized by the rapid elongation of stems and petioles, leaf hyponasty, reduced branching, and growth of the stems toward canopy gaps. All of these responses allow plants to forage for light in complex canopy environments [9,59].

Recent work has demonstrated that plants can adjust their investment in defense as a function of the perceived risk of competition. Physiological and genetic evidence indicates that the expression of the SAS is correlated with decreased expression of defense markers and increased vulnerability to insect herbivores [51,52,54]. The reduced investment in defense and increased tissue quality in plants exposed to low R:FR ratios, or in *phyB* mutants, is not a simple byproduct of the diversion of resources to shade-avoidance responses. This has been demonstrated in experiments involving the *sav3* mutant of *Arabidopsis*, where supplemental FR fails to elicit the SAS phenotype [60] but still downregulates plant defense [54].

The downregulation of defense in plants that confront an imminent risk of competition is mediated, at least in

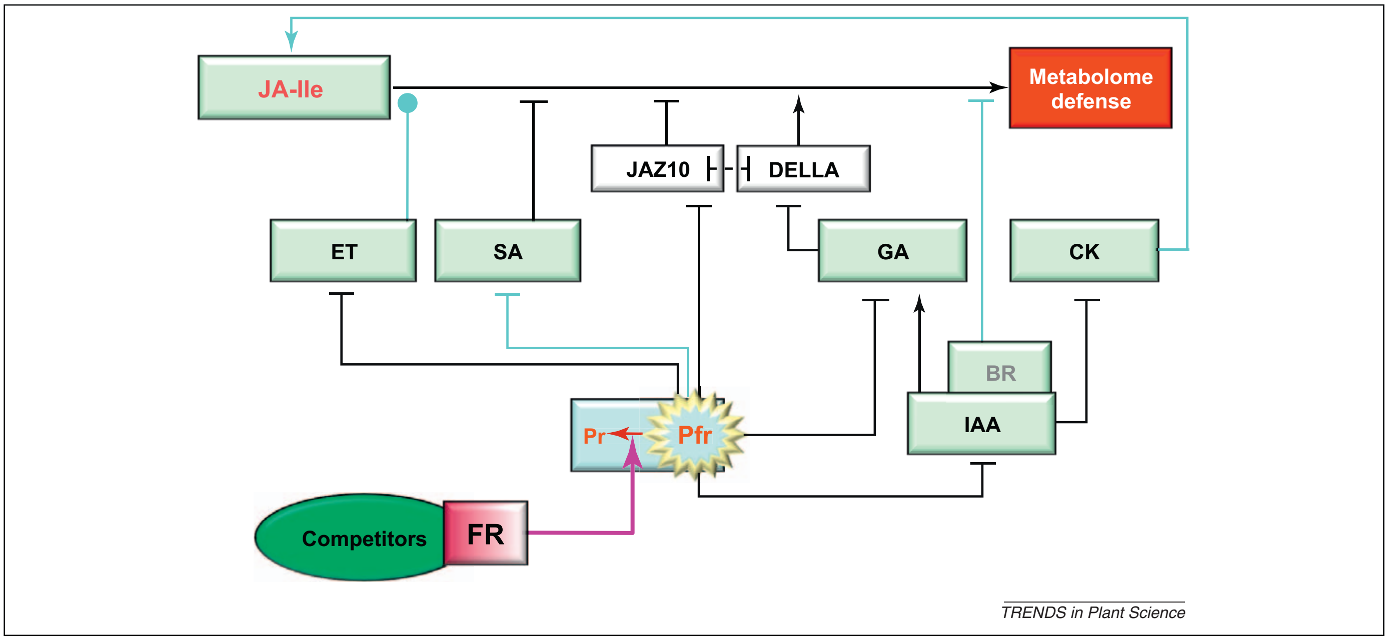

Figure 3. Potential connections between phyB and JA signaling in the modulation of defense responses by neighbor proximity. FR radiation, a signal of neighbor proximity in plant canopies, prompts the passage of the active, Pfr form of phyB into the inactive, Pr form. Reduced Pfr levels lead to the increased expression of some *JAZ* genes in JA-elicited tissues, which might depress the expression of JA responses under conditions of competition. The depletion of Pfr levels also leads to the increased action of several growth-promoting hormones, such as GA, BR, auxins and ET. GA and BR have been shown to repress JA responses in some systems. GAs act by promoting DELLA degradation, and very recent evidence suggests that DELLAs can directly interact with (and prevent the repressor activity of) JAZ1 (and presumably other JAZ) proteins [113]. ET has mixed, system-dependent effects. Increased IAA levels can increase CK turnover, and CKs have been shown to increase JA biosynthesis. SA, a repressor of JA action, can also be enhanced by Pfr depletion in some tissues and species. Arrows indicate promotion/activation; truncated lines indicate repression or negative interactions; connectors with a round point indicate system-dependent effects. Black lines denote interactions documented in *Arabidopsis*; pale blue lines denote interactions documented in other species. BR, brassinosteroid; CK, cytokinin; ET, ethylene; GA, gibberellin; IAA, indole-acetic acid (an auxin); SA, salicylate.

part, by a reduction in the sensitivity of plant tissues to JA [54]. The mechanisms whereby Pfr depletion leads to reduced JA sensitivity remain to be elucidated; some of the possibilities are depicted in Figure 3. JAZ proteins are negative regulators of JA-responsive genes (Figure 1) (reviewed in [1,17,18]) and are likely to play a role in attenuating JA sensitivity in response to physiological and environmental signals. A splice variant of JAZ10 (JAZ10.4) lacks the Jas domain (Figure 1) and does not interact with COI1 [61]. This isoform is therefore resistant to JA-induced degradation and, when ectopically overexpressed, causes dominant repression of JA responses [61]. The production of dominant JAZ repressors by alternative splicing has been hypothesized to be a general mechanism to reduce JA sensitivity [62]. A mechanism of this nature might be functionally significant in mediating the effects of phyB inactivation by canopy signals because phyB-Pfr depletion upregulates the expression of some *JAZ* genes, and increased transcript levels of *JAZ10* [54] and *JAZ10.4* (M. Keller and C. Ballaré, unpublished) have been found in response to low R:FR ratios. Interactions between phyA (another member of the phytochrome family) and JA in the control of growth responses during *Arabidopsis* seedling de-etiolation, mediated by JAZ1 stability, have recently been described; thus, the activation of phyA has been found to be required for COI1-mediated JAZ1 turnover [63]. Further work is needed to establish how this interaction plays out to regulate the effects of phyB-Pfr depletion on JA sensitivity in fully de-etiolated plants.

Many additional players might transduce the low phyB-Pfr signal to downregulate the output of the JA pathway. These include gibberellins (GAs), cytokinins (CKs), auxin, ET, brassinosteroids (BRs) and SA (Figure 3). GA levels increase in response to low R:FR ratios, causing the degradation of DELLA proteins [64]. Reduced levels of DELLA have been associated with a low sensitivity to JA and increased sensitivity to SA [65]. CKs regulate JA synthesis (see below) and are regulated by R:FR [66]. Low R:FR ratios result in increased auxin levels through the activation of the TAA1 pathway [60]. This change in auxin homeostasis might increase GA levels [67] and promote the breakdown of CKs [66], as discussed in [68]. Low R:FR ratios stimulate plants to produce ET [69], which is a well-known modulator with diverse effects on JA responses [7,25,43]. SAS induction by low R:FR has been connected with increased BR action in *Arabidopsis* [70], and BRs have been reported to antagonize JA in the induction of defense traits in the tomato [71]. An effect of low R:FR increasing SA levels has also been reported in some systems [72]; if this is a general effect, it could have consequences for JA responses, as discussed in the previous section. Finally, the *PFT1* gene (*PHYTOCHROME AND FLOWERING TIME 1*), which encodes a subunit of the Mediator complex (MED25) and is involved in the control of certain phytochrome responses [73], has recently been shown to play a role as a positive regulator of JA-induced defenses [74]. A precise map of the interactions among these putative modulators is still missing; however, the fact that so many (potentially redundant) control mechanisms seem to connect phytochromes with JA signaling suggests that this connection plays a central role in modulating resource allocation between growth and defense. Interestingly, recent work in the lima bean (*Phaseolus lunatus*) has suggested that phytochromes can also control the expression of indirect defenses (extrafloral nectar secretion) via the regulation of JA-Ile production and signaling [75].

In addition to R light, green leaves absorb strongly in the blue (B) and ultraviolet (UV) regions of the spectrum. Therefore, the light environment in dense canopies is also characterized by low levels of B and UV radiation. Plants have specific photoreceptors that are sensitive to these wavelengths, and low levels of B and UV radiation elicit SAS-like responses such as leaf hyponasty and stem elongation [9,58]. Solar UV-B radiation has well-known effects, making plants more resistant to insect herbivory [76,77], and it has been demonstrated that the expression of some of the antiherbivore effects of natural UV-B doses requires an intact JA signaling cascade [78,79]. UV-B radiation amplifies certain JA responses in *N. attenuata* (the accumulation of trypsin proteinase inhibitors) [79], which suggests that UV-B can act in the natural environment as a canopy signal that conveys the opposite information to that conveyed by low R:FR (i.e. a signal of high light and, therefore, no limitation to the investment of resources in antiherbivore defense). Whether the potentiating effect of UV-B shares molecular components with the phenomenon of 'priming' [80] by biotic signals (discussed below) remains to be determined.

Proximity effects on defense expression are not limited to total defense; allocation to different defense products can also be altered in the presence of neighboring plants [81]. Whether this effect of neighbor proximity is mediated by the regulation of JA responses is not known. In addition, the information that plants gather from neighboring plants is not limited to light signals. Plant-produced volatile compounds can convey important information on plant–herbivore interactions taking place in the neighborhood (reviewed in [27]), as discussed below.

Listening to internal signals and priming the defense response

The expression of the JA signaling pathway is also modulated by internal signals that might indicate the progression of the attack through the plant body or the value of the tissues to be defended. Two examples of those internal signals are levels of volatile organic compounds (VOCs) and CKs (Figure 2).

Many plants respond to insect herbivory or tissue damage by releasing blends of VOCs. Recent studies have shown that herbivory-elicited VOCs are involved in signaling within plants, complementing other mobile signals that are transported through the vasculature (reviewed in [27,82]). Herbivory-elicited VOCs can prime the production of defense responses in undamaged systemic tissues [83–85]. Plant receptors for herbivore-induced VOCs (with the exception of ET) have not been identified. Priming involves modifications in the transcriptome/metabolome that, while not conferring resistance *per se*, prepare the plant for a faster or more intense response to attack by herbivores or pathogens (the 'primed' state) [80].

Priming by volatile signals has been connected with the activation of the JA signaling pathway. For example, priming by fatty acid-derived green leaf volatiles (GLVs), a particular group of VOCs that are immediately (and probably passively) released from wounded leaves [86], has been shown to increase herbivore-induced JA synthesis and the expression of octadecanoid biosynthetic genes in maize (*Zea mays*) and hybrid poplar (*Populus deltoides* × *nigra*) [87–89] (Figure 2). In the poplar, the priming effect resulted in a stronger activation of direct and indirect defenses when the leaves were exposed to herbivory by gypsy moth (*Lymantria dispar*) larvae [89].

CKs are plant hormones involved in the regulation of many physiological processes, including branching, leaf growth, senescence and the distribution of N and photosynthetic capacity within the plant shoot. Higher levels of CKs are normally found in young, top canopy leaves. This is a consequence of a higher rate of synthesis and a faster rate of delivery of xylem-transported hormone to sun-exposed leaves with high transpiration rates [90,91]. Recent work with hybrid poplar has shown that CKs prime plant responses to wounding, reducing insect growth [92]. This priming effect of CKs, like the effect of GLVs, is also associated with increased JA biosynthesis and expression of herbivory-induced genes [92]. It is tempting to speculate, based on these results, that CKs provide a measure of the 'relative value' of a leaf and contribute to concentrating the expression of JA-induced defenses in those leaves that have the highest likelihood of contributing to the overall light capture and C balance of the plant.

The mechanisms whereby GLVs (or other VOCs) and CKs enhance JA production/response are not clear, but progress has been made in the identification of molecular players involved in priming by other regulatory signals (see section on ISR below).

The role of friends

The expression of JA-controlled defenses can be induced or primed by signals derived from 'friendly' neighboring plants and beneficial plant-associated microorganisms. As discussed in the previous section, branches attacked by herbivores produce VOCs that can prime other parts of the same plant for a stronger defense response. Depending on the characteristics of the VOCs released by damaged tissues, the size of the emissions and the aerodynamic characteristics of the whole canopy, these volatiles could reach the shoots of neighboring plants and act as warning signals of oncoming herbivore attacks (reviewed in [27,93]). The history of the field and the range of species in which experimental evidence has shown that airborne compounds produced by induced plants trigger antiherbivore resistance or the expression of resistance-related markers in intact plants is discussed in [27]. The evolutionary significance of this type of interplant communication remains unclear [27,93]. Interestingly, evidence from a field study with sagebrush (*Artemisia tridentata*) suggests that communication between 'emitter' and 'receiver' plants is favored by genetic relatedness [94]. In addition, a recent study with *Centaurea maculosa* has suggested that the effect of JA, eliciting plant defenses, varied depending on the plant species planted next to the treated plant; the nature of the signal that conveyed species-specific information was not investigated [95].

Beneficial soil microorganisms (such as mycorrhizae and certain rhizobacteria) can also induce a primed state in aboveground plant organs, which provides enhanced protection against a broad spectrum of pathogens and insect herbivores [96–98]. This particular priming phenomenon has been called induced systemic resistance (ISR) [7] and is characterized by a faster and stronger expression of JA-related defense responses when the primed organs are attacked by pathogens or insects (Figure 2). Similar to the case of priming induced by VOCs, the molecular mechanisms that increase the JA response in the target tissues have not been established, but several clues have emerged from recent work with *Arabidopsis* plants colonized by the plant growth-promoting rhizobacteria *Pseudomonas fluorescens* WCS417r [97,99]. MYC2 and AP2/ERF transcription factors (which are key players in JA-induced responses [100]) are emerging as important regulators in priming during ISR (reviewed in [97]). Although *P. fluorescens* WCS417r can cause (modest) increases in the expression of *MYC2* and other transcription factor genes, no significant activation of target genes was observed until the plants were actually infected by a pathogen [101]. This observation has been interpreted [97] as preliminary evidence for a model of the primed state in which transcription factors remain inactive (e.g. in the cytosol) until the plant is effectively attacked by a JA-inducing insect or pathogen. Therefore, according to this model, regulatory events that act at the post-translational level are required for the priming mechanism [97].

The rascals

No defense system is perfect and several groups of consumer organisms have evolved crafty mechanisms to foul JA responses. As explained above, SA is a well-known depressor of JA-induced defenses [7,25,28]. Accordingly, application of SA and inoculation with SA-inducing pathogens sometimes reduce plant resistance to insect herbivores [102–104]. Studies in *Arabidopsis* and *N. attenuata* have suggested that generalist herbivores such as *Spodoptera* spp. can activate (through an unidentified mechanism) the SA pathway during feeding, which might help them attenuate JA-mediated defense responses mounted by the host plant [42,103–106]. Similar decoy tactics are used by nymphs of the phloem-feeding silverleaf whitefly (*Bremia tabaci*) [107]. The suppression of JA responses by elicitors present in insect eggs, which activate the SA pathway and locally suppress plant defenses at the site of oviposition, represents an exquisite example of the manipulation of plant hormonal crosstalk to the advantage of the consumer organism [40]. Other examples of consumer-induced downregulation of JA responses and concomitant weakening of plant defenses involve the elicitation of an ET burst by the specialist herbivore *Manduca sexta* [48] and the production of JA-antagonistic GA by the necrotrophic fungus *Gibberella fujikuroi* [65].

Concluding remarks

The JA signaling pathway represents a central regulator of inducible plant defenses. Major progress has been made in the past five years in the elucidation of the mechanisms of JA perception (Figure 1). In addition, a combination of

Box 2. Outstanding questions

- Little is known about the processes that operate upstream of JA biosynthesis [5]. Do ecological modulators also affect HAMP/DAMP perception and the extent to which the JA response is triggered by these signals?
- Our understanding of the functional connections between the ecological modulators described in this review and the early events of JA signaling (Figures 2 and 3) is still notoriously underdeveloped. Why do plants have multiple *JAZ* genes (12 in *Arabidopsis*) and what is the map of interactions between the resulting proteins and the key transcription factors? What are the physiological roles of homo- and heteromeric protein–protein interactions among JAZ proteins [61]? Most JAZ genes in *Arabidopsis* seem to be subject to alternative splicing events within the Jas domain, which generate transcripts encoding truncated JAZ proteins. These truncated JAZs have a reduced capacity to form complexes with COI1 and have been proposed to play an important role as dominant repressors of JA responses [62]. However, the functional roles of these JAZ splice variants in the ecological modulation of JA signaling have yet to be demonstrated.
- Do physiological and ecological signals lead to the production of different (specialized?) JA coreceptor complexes (Figure 1), with different properties and signaling functions, by modulating the relative abundance of the various JAZ proteins and splice variants?
- Besides the regulation that takes place at the COI1-JAZ module, what is the relative importance of the downstream controls, such as those proposed to regulate some of the SA effects [39]?
- Do the various signals potentially connected with phyB inactivation (Figure 3) play a functional role? Do they converge at a common point in JA signaling? In the same vein, is there a more-or-less general mechanism of priming, which could account for the positive effects on JA sensitivity of biotic signals (ISR, VOCs, etc.) and abiotic stressors (UV)?
- How are the various crosstalk mechanisms outlined here interlocked and functionally connected, and how do they contribute to shape the defense phenotype under natural conditions? Advances in the field of systems biology combined with the use of multiple mutants could help visualize the output of interacting signaling networks [110].
- What is the spatial variation in the expression of JA responses throughout the plant body? Light and hormonal signals might tag particular organs as ‘dispensable’ or ‘valuable’, and regulate the JA response accordingly (Figure 2). Similarly, hormonal antagonism (e.g. SA–JA) can be restricted to certain parts of the plant body [30]. Further work is needed to understand how signaling crosstalk operates at the whole plant level.
- JA-induced defense responses are modulated by plant density [54], UV radiation [79], atmospheric CO_2 [111] and possibly air pollutants [112]. How will the expression of JA responses be affected by agricultural intensification (increased crop density and breeding for higher biomass yield) and climate change factors predicted for the coming decades?

molecular ecology and genetic approaches is producing significant advances in our understanding of the mechanisms that modulate JA responses and control the plastic expression of plant defenses (Figures 2 and 3). This progress has revealed novel, unexpected informational inputs to the JA pathway, including those produced by changes in the phytochrome status (caused by the proximity of neighboring plants), and variations in the levels of growth-related hormones such as GAs, BRs and CKs. In addition, novel approaches have provided new insights into previously established interactions, such as those that underlie the effects of biotrophic pathogens and beneficial microorganisms on JA signaling. This improved understanding is beginning to generate coarse maps of the signaling circuits that define the outcomes of intelligent plant defense decisions in the natural environment. Several exciting new questions have emerged (Box 2), which will probably motivate future research in this fast-moving field.

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References

1 Browse, J. (2009) Jasmonate passes muster: a receptor and targets for the defense hormone. *Annu. Rev. Plant Biol.* 60, 183–205
2 Farmer, E.E. and Ryan, C.A. (1990) Interplant communication: airborne methyl jasmonate induces synthesis of proteinase inhibitors in plant leaves. *Proc. Natl. Acad. Sci. U.S.A.* 87, 7713–7716
3 Farmer, E.E. and Ryan, C.A. (1992) Octadecanoid precursors of jasmonic acid activate the synthesis of wound-inducible proteinase inhibitors. *Plant Cell* 4, 129–134
4 Felton, G.W. and Tumlinson, J.H. (2008) Plant-insect dialogs: complex interactions at the plant-insect interface. *Curr. Opin. Plant Biol.* 11, 457–463
5 Howe, G.A. and Jander, G. (2008) Plant immunity to insect herbivores. *Annu. Rev. Plant Biol.* 59, 41–66
6 Kazan, K. and Manners, J.M. (2008) Jasmonate signaling: toward an integrated view. *Plant Physiol.* 146, 1459–1468
7 Pieterse, C.M.J. *et al.* (2009) Networking by small-molecule hormones in plant immunity. *Nat. Chem. Biol.* 5, 308–316
8 Verhage, A. *et al.* (2010) Plant immunity: it’s the hormones talking, but what do they say? *Plant Physiol.* 154, 536–540
9 Ballaré, C.L. (2009) Illuminated behaviour: phytochrome as a key regulator of light foraging and plant anti-herbivore defence. *Plant Cell Environ.* 32, 713–725
10 Chini, A. *et al.* (2007) The JAZ family of repressors is the missing link in jasmonate signalling. *Nature* 448, 666–671
11 Thines, B. *et al.* (2007) JAZ repressor proteins are targets of the SCF^{COI1} complex during jasmonate signalling. *Nature* 448, 661–665
12 Yan, Y. *et al.* (2007) A downstream mediator in the growth repression limb of the jasmonate pathway. *Plant Cell* 19, 2470–2483
13 Melotto, M. *et al.* (2008) A critical role of two positively charged amino acids in the Jas motif of *Arabidopsis* JAZ proteins in mediating coronatine- and jasmonoyl isoleucine-dependent interactions with the COI1 F-box protein. *Plant J.* 55, 979–988
14 Yan, J.B. *et al.* (2009) The *Arabidopsis* CORONATINE INSENSITIVE1 protein is a jasmonate receptor. *Plant Cell* 21, 2220–2236
15 Pauwels, L. *et al.* (2010) NINJA connects the co-repressor TOPLESS to jasmonate signalling. *Nature* 464, 788–791
16 Sheard, L.B. *et al.* (2010) Jasmonate perception by inositol-phosphate-potentiated COI1-JAZ co-receptor. *Nature* 468, 400–407
17 Fonseca, S. *et al.* (2009) The jasmonate pathway: the ligand, the receptor and the core signalling module. *Curr. Opin. Plant Biol.* 12, 539–547
18 Chung, H.S. *et al.* (2009) Top hits in contemporary JAZ: an update on jasmonate signaling. *Phytochem.* 70, 1547–1559
19 Howe, G.A. (2010) Ubiquitin ligase-coupled receptors extend their reach to jasmonate. *Plant Physiol.* 154, 471–474
20 Koo, A.J.K. *et al.* (2009) A rapid wound signal activates the systemic synthesis of bioactive jasmonates in *Arabidopsis*. *Plant J.* 59, 974–986
21 Karban, R. (2008) Plant behaviour and communication. *Ecol. Lett.* 11, 727–739
22 Trewavas, A. (2009) What is plant behaviour? *Plant Cell Environ.* 32, 606–616
23 Cipollini, D. (2004) Stretching the limits of plasticity: can a plant defend against both competitors and herbivores? *Ecology* 85, 28–37

24 Baldwin, I.T. (2001) An ecologically motivated analysis of plant-herbivore interactions in native tobacco. *Plant Physiol.* 127, 1449–1458
25 Bostock, R.M. (2005) Signal crosstalk and induced resistance: straddling the line between cost and benefit. *Annu. Rev. Phytopathol.* 43, 545–580
26 Cipollini, D. (2010) Constitutive expression of methyl jasmonate-inducible responses delays reproduction and constrains fitness responses to nutrients in *Arabidopsis thaliana*. *Evol. Ecol.* 24, 59–68
27 Heil, M. and Karban, R. (2010) Explaining evolution of plant communication by airborne signals. *Trends Ecol. Evol.* 25, 137–144
28 Kunkel, B.N. and Brooks, D.M. (2002) Cross talk between signaling pathways in pathogen defense. *Curr. Opin. Plant Biol.* 5, 325–331
29 Koornneef, A. and Pieterse, C.M.J. (2008) Cross talk in defense signaling. *Plant Physiol.* 146, 839–844
30 Spoel, S.H. and Dong, X. (2008) Making sense of hormone crosstalk during plant immune responses. *Cell Host Microb.* 3, 348–351
31 Felton, G.W. and Korth, K.L. (2000) Trade-offs between pathogen and herbivore resistance. *Curr. Opin. Plant Biol.* 3, 309–314
32 Spoel, S.H. *et al.* (2007) Regulation of tradeoffs between plant defenses against pathogens with different lifestyles. *Proc. Natl. Acad. Sci. U.S.A.* 104, 18842–18847
33 Brooks, D.M. *et al.* (2005) The *Pseudomonas syringae* phytotoxin coronatine promotes virulence by overcoming salicylic acid-dependent defences in *Arabidopsis thaliana*. *Mol. Plant Pathol.* 6, 629–639
34 Uppalapati, S.R. *et al.* (2007) The phytotoxin coronatine contributes to pathogen fitness and is required for suppression of salicylic acid accumulation in tomato inoculated with *Pseudomonas syringae* pv. tomato DC3000. *Mol. Plant-Microbe Interac.* 20, 955–965
35 Spoel, S.H. *et al.* (2003) NPR1 modulates cross-talk between salicylate- and jasmonate-dependent defense pathways through a novel function in the cytosol. *Plant Cell* 15, 760–770
36 Leon-Reyes, A. *et al.* (2010) Salicylate-mediated suppression of jasmonate-responsive gene expression in *Arabidopsis* is targeted downstream of the jasmonate biosynthesis pathway. *Planta* 232, 1423–1432
37 Dong, X. (2004) NPR1, all things considered. *Curr. Opin. Plant Biol.* 7, 547–552
38 Glazebrook, J. *et al.* (2003) Topology of the network integrating salicylate and jasmonate signal transduction derived from global expression phenotyping. *Plant J.* 34, 217–228
39 Leon-Reyes, A. (2009) Making sense out of signaling during plant defense. Doctoral Thesis. Utrecht University
40 Bruessow, F. *et al.* (2010) Insect eggs suppress plant defence against chewing herbivores. *Plant J.* 62, 876–885
41 Leon-Reyes, A. *et al.* (2009) Ethylene modulates the role of NONEXPRESSOR OF PATHOGENESIS-RELATED GENES1 in cross talk between salicylate and jasmonate signaling. *Plant Physiol.* 149, 1797–1809
42 Rayapuram, C. and Baldwin, I.T. (2007) Increased SA in NPR1-silenced plants antagonizes JA and JA-dependent direct and indirect defenses in herbivore-attacked *Nicotiana attenuata* in nature. *Plant J.* 52, 700–715
43 von Dahl, C. and Baldwin, I. (2007) Deciphering the role of ethylene in plant–herbivore interactions. *J. Plant Growth Reg.* 26, 201–209
44 Penninckx, I.A.M.A. *et al.* (1998) Concomitant activation of Jasmonate and ethylene response pathways is required for induction of a plant defensin gene in *Arabidopsis*. *Plant Cell* 10, 2103–2114
45 Lorenzo, O. *et al.* (2003) ETHYLENE RESPONSE FACTOR1 integrates signals from ethylene and jasmonate pathways in plant defense. *Plant Cell* 15, 165–178
46 Pre, M. *et al.* (2008) The AP2/ERF domain transcription factor ORA59 integrates jasmonic acid and ethylene signals in plant defense. *Plant Physiol.* 147, 1347–1357
47 Manavella, P.A. *et al.* (2008) HAHB4, a sunflower HD-Zip protein, integrates signals from the jasmonic acid and ethylene pathways during wounding and biotic stress responses. *Plant J.* 56, 376–388
48 Winz, R.A. and Baldwin, I.T. (2001) Molecular interactions between the specialist herbivore *Manduca sexta* (Lepidoptera, Sphingidae) and its natural host *Nicotiana attenuata*. IV. Insect-induced ethylene reduces jasmonate-induced nicotine accumulation by regulating putrescine N-methyltransferase transcripts. *Plant Physiol.* 125, 2189–2202
49 Leon-Reyes, A. *et al.* (2010) Ethylene signaling renders the jasmonate response of *Arabidopsis* insensitive to future suppression by salicylic acid. *Mol. Plant-Microbe Interac.* 23, 187–197
50 De Vos, M. *et al.* (2005) Signal signature and transcriptome changes of *Arabidopsis* during pathogen and insect attack. *Mol. Plant-Microbe Interac.* 18, 923–937
51 McGuire, R. and Agrawal, A.A. (2005) Trade-offs between the shade-avoidance response and plant resistance to herbivores? Tests with mutant *Cucumis sativus*. *Funct. Ecol.* 19, 1025–1031
52 Izaguirre, M.M. *et al.* (2006) Remote sensing of future competitors: impacts on plant defenses. *Proc. Natl. Acad. Sci. U.S.A.* 103, 7170–7174
53 Baldwin, I.T. (1998) Jasmonate induced responses are costly but benefit plants under attack in native populations. *Proc. Natl. Acad. Sci. U.S.A.* 95, 8113–8118
54 Moreno, J.E. *et al.* (2009) Ecological modulation of plant defense via phytochrome control of jasmonate sensitivity. *Proc. Natl. Acad. Sci. U.S.A.* 106, 4935–4940
55 Herms, D.A. and Mattson, W.J. (1992) The dilemma of plants: to grow or defend. *Quart. Rev. Biol.* 67, 283–335
56 Franklin, K.A. and Quail, P.H. (2010) Phytochrome functions in *Arabidopsis* development. *J. Expt. Bot.* 61, 11–24
57 Kami, C. *et al.* (2010) Light-regulated plant growth and development. In *Current Topics in Developmental Biology*, pp. 29–66, Academic Press
58 Keuskamp, D.H. *et al.* (2010) Physiological regulation and functional significance of shade avoidance responses to neighbors. *Plant Signal. Behav.* 5, 1–8
59 Schmitt, J. *et al.* (2003) The adaptive evolution of plasticity: phytochrome-mediated shade avoidance responses. *Integr. Comp. Biol.* 43, 459–469
60 Tao, Y. *et al.* (2008) Rapid synthesis of auxin via a new tryptophan-dependent pathway is required for shade avoidance in plants. *Cell* 133, 164–176
61 Chung, H.S. and Howe, G.A. (2009) A critical role for the TIFY motif in repression of jasmonate signaling by a stabilized splice variant of the JASMONATE ZIM-domain protein JAZ10 in *Arabidopsis*. *Plant Cell* 21, 131–145
62 Chung, H.S. *et al.* (2010) Alternative splicing expands the repertoire of dominant JAZ repressors of jasmonate signaling. *Plant J.* 63, 613–622
63 Robson, F. *et al.* (2010) Jasmonate and phytochrome a signaling in *Arabidopsis* wound and shade responses are integrated through JAZ1 stability. *Plant Cell* 22, 1143–1160
64 Djakovic-Petrovic, T. *et al.* (2007) DELLA protein function in growth responses to canopy signals. *Plant J.* 51, 117–126
65 Navarro, L. *et al.* (2008) DELLAs control plant immune responses by modulating the balance of jasmonic acid and salicylic acid signaling. *Curr. Biol.* 18, 650–655
66 Carabelli, M. *et al.* (2007) Canopy shade causes a rapid and transient arrest in leaf development through auxin-induced cytokinin oxidase activity. *Genes Dev.* 21, 1863–1868
67 Frigerio, M. *et al.* (2006) Transcriptional regulation of gibberellin metabolism genes by auxin signaling in *Arabidopsis*. *Plant Physiol.* 142, 553–563
68 Jaillais, Y. and Chory, J. (2010) Unraveling the paradoxes of plant hormone signaling integration. *Nat. Struct. Mol. Biol.* 17, 642–645
69 Kegge, W. and Pierik, R. (2010) Biogenic volatile organic compounds and plant competition. *Trends Plant Sci.* 15, 126–132
70 Kozuka, T. *et al.* (2010) Involvement of auxin and brassinosteroid in the regulation of petiole elongation under the shade. *Plant Physiol.* 153, 1608–1618
71 Campos, M.L. *et al.* (2009) Brassinosteroids interact negatively with jasmonates in the formation of anti-herbivory traits in tomato. *J. Exp. Bot.* 60, 4347–4361
72 Kurepin, L.V. *et al.* (2010) Light regulation of endogenous salicylic acid levels in hypocotyls of *Helianthus annuus* seedlings. *Botany* 88, 668–674
73 Wollenberg, A.C. *et al.* (2008) Acceleration of flowering during shade avoidance in *Arabidopsis* alters the balance between Flowering Locus C-mediated repression and photoperiodic induction of flowering. *Plant Physiol.* 148, 1681–1694

74 Kidd, B.N. *et al.* (2009) The mediator complex subunit PFT1 is a key regulator of jasmonate-dependent defense in *Arabidopsis*. *Plant Cell* 21, 2237–2252
75 Radhika, V. *et al.* (2010) Regulation of extrafloral nectar secretion by jasmonates in lima bean is light dependent. *Proc. Natl. Acad. Sci. U.S.A.* 107, 17228–17233
76 Ballaré, C.L. *et al.* (1996) Solar ultraviolet-B radiation affects seedling emergence, DNA integrity, plant morphology, growth rate, and attractiveness to herbivore insects in *Datura ferox*. *Plant Physiol.* 112, 161–170
77 Mazza, C.A. *et al.* (1999) Perception of solar UVB radiation by phytophagous insects: behavioral responses and ecosystem implications. *Proc. Natl. Acad. Sci. U.S.A.* 96, 980–985
78 Caputo, C. *et al.* (2006) Solar ultraviolet-B radiation alters the attractiveness of *Arabidopsis* plants to diamondback moths (*Plutella xylostella* L.): impacts on oviposition and involvement of the jasmonic acid pathway. *Oecologia* 149, 81–90
79 Demkura, P.V. *et al.* (2010) Jasmonate-dependent and -independent pathways mediate specific effects of solar ultraviolet-B radiation on leaf phenolics and antiherbivore defense. *Plant Physiol.* 152, 1084–1095
80 Conrath, U. *et al.* (2006) Priming: getting ready for battle. *Mol. Plant-Microbe Interac.* 19, 1062–1071
81 Wentzell, A.M. and Kliebenstein, D.J. (2008) Genotype, age, tissue, and environment regulate the structural outcome of glucosinolate activation. *Plant Physiol.* 147, 415–428
82 Frost, C.J. *et al.* (2008) Plant defense priming against herbivores: getting ready for a different battle. *Plant Physiol.* 146, 818–824
83 Karban, R. *et al.* (2006) Damage-induced resistance in sagebrush: volatiles are key to intra- and interplant communication. *Ecology* 87, 922–930
84 Frost, C.J. *et al.* (2007) Within-plant signalling via volatiles overcomes vascular constraints on systemic signalling and primes responses against herbivores. *Ecol. Lett.* 10, 490–498
85 Heil, M. and Bueno, J.C.S. (2007) Within-plant signaling by volatiles leads to induction and priming of an indirect plant defense in nature. *Proc. Natl. Acad. Sci. U.S.A.* 104, 5467–5472
86 D'Auria, J.C. *et al.* (2007) Characterization of a BAHD acyltransferase responsible for producing the green leaf volatile (Z)-3-hexen-1-yl acetate in *Arabidopsis thaliana*. *Plant J.* 49, 194–207
87 Engelberth, J. *et al.* (2004) Airborne signals prime plants against insect herbivore attack. *Proc. Natl. Acad. Sci. U.S.A.* 101, 1781–1785
88 Engelberth, J. *et al.* (2007) Insect elicitors and exposure to green leafy volatiles differentially upregulate major octadecanoids and transcripts of 12-oxo phytodienoic acid reductases in *Zea mays*. *Mol. Plant-Microbe Interac.* 20, 707–716
89 Frost, C.J. *et al.* (2008) Priming defense genes and metabolites in hybrid poplar by the green leaf volatile cis-3-hexenyl acetate. *New Phytol.* 180, 722–734
90 Boonman, A. *et al.* (2007) Cytokinin import rate as a signal for photosynthetic acclimation to canopy light gradients. *Plant Physiol.* 143, 1841–1852
91 Boonman, A. *et al.* (2009) Redundant roles of photoreceptors and cytokinins in regulating photosynthetic acclimation to canopy density. *J. Expt. Bot.* 60, 1179–1190
92 Dervinis, C. *et al.* (2010) Cytokinin primes plant responses to wounding and reduces Insect performance. *J. Plant Growth Reg.* 29, 289–296
93 Dicke, M. and Baldwin, I.T. (2010) The evolutionary context for herbivore-induced plant volatiles: beyond the 'cry for help'. *Trends Plant Sci.* 15, 167–175
94 Karban, R. and Shiojiri, K. (2009) Self-recognition affects plant communication and defense. *Ecol. Lett.* 12, 502–506
95 Broz, A.K. *et al.* (2010) Plant neighbor identity influences plant biochemistry and physiology related to defense. *BMC Plant Biol.* 10, 115
96 Van Wees, S.C.M. *et al.* (2008) Plant immune responses triggered by beneficial microbes. *Curr. Opin. Plant Biol.* 11, 443–448
97 Van der Ent, S. *et al.* (2009) Jasmonate signaling in plant interactions with resistance-inducing beneficial microbes. *Phytochem.* 70, 1581–1588
98 Pineda, A. *et al.* (2010) Helping plants to deal with insects: the role of beneficial soil-borne microbes. *Trends Plant Sci.* 15, 507–514
99 Pozo, M.J. *et al.* (2008) Transcription factor MYC2 is involved in priming for enhanced defense during rhizobacteria-induced systemic resistance in *Arabidopsis thaliana*. *New Phytol.* 180, 511–523
100 Lorenzo, O. and Solano, R. (2005) Molecular players regulating the jasmonate signalling network. *Curr. Opin. Plant Biol.* 8, 532–540
101 Van der Ent, S. *et al.* (2009) Priming of plant innate immunity by rhizobacteria and beta-aminobutyric acid: differences and similarities in regulation. *New Phytol.* 183, 419–431
102 Preston, C.A. *et al.* (1999) Tobacco mosaic virus inoculation inhibits wound-induced jasmonic acid-mediated responses within but not between plants. *Planta* 209, 87–95
103 Stotz, H.U. *et al.* (2002) Evidence for regulation of resistance in *Arabidopsis* to Egyptian cotton worm by salicylic and jasmonic acid signaling pathways. *Planta* 214, 648–652
104 Cipollini, D. *et al.* (2004) Salicylic acid inhibits jasmonic acid-induced resistance of *Arabidopsis thaliana* to *Spodoptera exigua*. *Mol. Ecol.* 13, 1643–1653
105 Weech, M.H. *et al.* (2008) Caterpillar saliva interferes with induced *Arabidopsis thaliana* defence responses via the systemic acquired resistance pathway. *J. Expt. Bot.* 59, 2437–2448
106 Diezel, C. *et al.* (2009) Different lepidopteran elicitors account for cross-talk in herbivory-induced phytohormone signaling. *Plant Physiol.* 150, 1576–1586
107 Zarate, S.I. *et al.* (2006) Silverleaf whitefly induces salicylic acid defenses and suppresses effectual jasmonic acid defenses. *Plant Physiol.* 143, 866–875
108 Glazebrook, J. (2005) Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. *Annu. Rev. Phytopathol.* 43, 205–227
109 Schoonhoven, L.M. *et al.* (2005) *Insect-Plant Biology*, Oxford University Press
110 Tsuda, K. *et al.* (2009) Network properties of robust immunity in plants. *PLoS Genetics* 5, e1000772
111 Zavala, J.A. *et al.* (2008) Anthropogenic increase in carbon dioxide compromises plant defense against invasive insects. *Proc. Natl. Acad. Sci. U.S.A.* 105, 5129–5133
112 Blande, J.D. *et al.* (2010) Air pollution impedes plant-to-plant communication by volatiles. *Ecol. Lett.* 13, 1172–1181
113 Hou, X. *et al.* (2010) DELLAs modulate jasmonate signaling via competitive binding to JAZs. *Dev. Cell* 19, 884–894