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REVIEW PAPER

The interplay between light and jasmonate signalling during defence and development

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Abstract

During their evolution, plants have acquired diverse capabilities to sense their environment and modify their growth and development as required. The versatile utilization of solar radiation for photosynthesis as well as a signal to coordinate developmental responses to the environment is an excellent example of such a capability. Specific light quality inputs are converted to developmental outputs mainly through hormonal signalling pathways. Accordingly, extensive interactions between light and the signalling pathways of every known plant hormone have been uncovered in recent years. One such interaction that has received recent attention and forms the focus of this review occurs between light and the signalling pathway of the jasmonate hormone with roles in regulating plant defence and development. Here the recent research that revealed new mechanistic insights into how plants might integrate light and jasmonate signals to modify their growth and development, especially when defending themselves from either pests, pathogens, or encroaching neighbours, is discussed.

Key words: *Arabidopsis*, COI1, *Fusarium*, MYC2, JAZ, PFT1, phytochrome, shade avoidance syndrome.

Introduction

Despite their sessile growth habits and rather rigid appearances, plants are extremely plastic creatures and adapt their environment remarkably well. Amazed with such elaborate plasticity displayed by plants, Edward Steichen (1879–1973), a photographer and a keen observer of nature, wrote 'I knew, of course, that trees and plants had roots, stems, bark, branches and foliage that reached up toward the light. But I was coming to realize that the real magician was light itself.' Indeed, solar radiation is one of the most important factors required for plant growth and development. Developmental plasticity in a given environment is achieved at least partly by constant monitoring of the quality, quantity, and direction of solar radiation. The capture of light energy by photosystems I and II in the chloroplast provides the energy for photosynthetic carbon fixation and biomass production. In addition to capture light for photosynthesis, plants have developed intricate means for the perception of specific light qualities and are able to transmit these signals to activate developmental programmes. This capability enables the plant to benefit optimally from the incident light.

Despite the significant progress made during the last two decades, our basic understanding of molecular processes involved in light perception and signalling is continually evolving. Importantly, it is becoming increasingly clear that, upon perception, light signals are skilfully integrated into other downstream signalling networks. In particular, plant hormone signalling pathways play important roles in converting light inputs into outputs that shape plant growth and development. For instance, light-mediated inhibition of hypocotyl elongation is at least in part mediated by the plant hormone gibberellin (GA). Another light-regulated developmental plant response, the shade avoidance syndrome (SAS), is primarily mediated by the plant hormone auxin, but also by other plant hormones such as brassinosteroids, cytokinins, GAs, and ethylene (reviewed by Wolters and Jürgens, 2009). Recent research also implicates the plant hormone jasmonate (JA) in a number of light-mediated responses, including SAS. In this paper, recent studies that have uncovered new integrative hubs for light and JA signalling are briefly reviewed.

Perception of light quality and signal transmission

Given the vital importance of light for their survival, plants have developed extremely sensitive and accurate capacities to sense different light spectra [red (R), far-red (FR), white, blue, green, and ultraviolet (UV)] present in solar radiation through the action of multiple photoreceptors. In *Arabidopsis*, the photoreceptor phytochrome proteins are encoded by five structurally different genes (PHYA–PHYE) and act as receptors for R and FR light with overlapping roles. Of these, PHYA is the main photoreceptor for FR light (700–800 nm) while white light and R light (700 nm) are primarily sensed by PHYB. In *Arabidopsis*, blue (~400 nm) and green (500–600 nm) lights are sensed by cryptochromes encoded by *CRY1* and *CRY2* (reviewed by Folta and Maruhnich, 2007; Jiao *et al.*, 2007; Bae and Choi, 2008). Solar radiation also contains various UV lights such as UV-A (320–390 nm), UV-B (280–315 nm), and UV-C (>280 nm). Of these, most UV-C and some UV-B radiation is captured by the ozone layer in the Stratosphere. Cryptochromes are also involved in UV-A sensing, but the nature of the UV-B receptor is currently unknown (reviewed by Jenkins, 2009).

When grown under FR light or in the dark, *Arabidopsis* seedlings display an 'etiolation' phenotype with elongated hypocotyls relative to their light-grown counterparts. This response is known as 'skotomorphogenesis'. In contrast, R light inhibits hypocotyl elongation and this response is known as 'photomorphogenesis'. This differential response of young seedlings to different light spectra has been instrumental in genetically assigning functional roles for different phytochromes in regulating light responses. For instance, the *phyA* mutant is compromised in seedling de-etiolation under continuous FR (cFR) light, indicating that PHYA acts as a negative regulator of skotomorphogenesis, while the *phyB* mutant displays a constitutive etiolation phenotype, indicating that PHYB is a positive regulator of photomorphogenesis (Jiao *et al.*, 2007; Martínez-García *et al.*, 2010).

Molecular mechanisms involved in light signalling have recently been extensively reviewed (Jiao *et al.*, 2007; Alabadí and Blázquez, 2009; Chory, 2010; Kami *et al.*, 2010; Lau and Deng, 2010). Briefly, in the dark, phytochromes repress light responses by physically interacting with PIFs (phytochrome-interacting factors), negative regulators of light responses. PIFs are members of the basic helix–loop–helix (bHLH) transcription factor gene family and bind to the G-box DNA sequence motif present in various light-regulated gene promoters. In addition, in the dark, positive regulators of light signalling such as HFR1 (LONG HYPOCOTYL IN FAR-RED1), HY5 (LONG HYPOCOTYL5), and LAF1 (LONG AFTER FAR-RED LIGHT1) are continuously degraded in the nucleus through the action of COP1 (CONSTITUTIVE PHOTOMORPHOGENIC1), a RING-finger-type ubiquitin E3 ligase that acts as a repressor of light signalling. Upon exposure to light, phytochromes move to the nucleus and negative regulators of light signalling (e.g. PIFs) are removed by the 26S proteasome. In addition, dark-mediated degradation of positive regulators by COP1 is inhibited under light by exclusion of COP1 from the nucleus, leading to the activation of light responses or photomorphogenesis.

Photoreceptors are also involved in detecting the quality of light by monitoring R:FR ratios. Phytochromes are synthesized in an R light-absorbing state known as 'Pr'. Upon excitation by R light, phytochromes are converted into the FR light-absorbing and biologically active 'Pfr' state (Fig. 1). Because R light is absorbed by plant pigments such as chlorophyll and carotenoid, its amount can be substantially reduced while passing through a dense canopy. A low R:FR ratio (<1) signals for the presence of potential competitors. Shade-intolerant plant species such as *Arabidopsis* respond to this potential threat by increasing stem elongation and accelerating flowering. This evolutionary phenomenon is known as the SAS (Fig. 1). PHYA and PHYB are both involved in SAS. PHYB inhibits SAS in

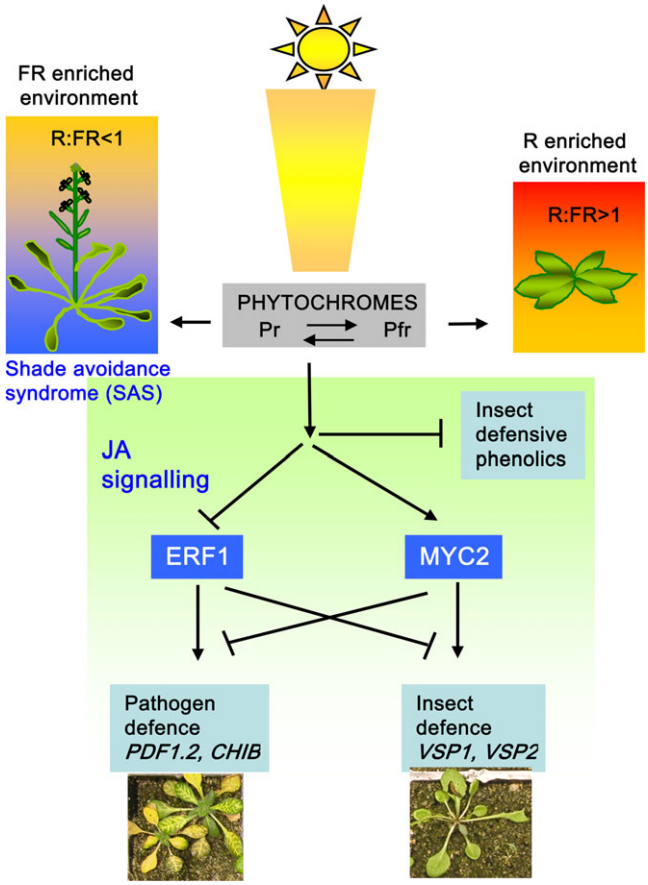

Fig. 1. Light quality affects both defence and development. FR light-enriched environments (R:FR <1) promote shade avoidance syndrome in shade-intolerant species such as *Arabidopsis*. In this model, FR light appears to regulate different JA-dependent responses differentially. FR light represses JA-responsive fungal defence genes such as *PDF1.2* through transcriptional repression of the JA-responsive AP2/ERF transcription factor *ERF1*. FR also represses the biosynthesis of JA-responsive insect defence compounds, leaf phenolics. In contrast, FR light activates the transcription from a subset of insect defence genes such as *VSP1* and *VSP2* through activation of the basic helix–loop–helix transcription factor MYC2. See the text for additional details.

R light-enriched conditions (R:FR >1) and *phyB* mutant plants display a constitutive SAS response. In contrast, PHYA inhibits SAS in FR light-enriched conditions (R:FR <1) (reviewed by Franklin, 2008; Lorrain *et al.*, 2008; Franklin and Quail, 2010; Jaillais and Chory, 2010; Martínez-García *et al.*, 2010; Stamm and Kumar, 2010).

Perception and transmission of JA signals

JA regulates plant pathogen and insect defence, wound responses, and diverse developmental processes. Biochemical events involved in JA biosynthesis have recently been reviewed (Wasternack, 2007; Wasternack and Kombrink, 2010).

Our understanding of molecular events associated with sensing of JA signals has recently been greatly improved with the discovery of a family of proteins called JAZ (JASMONATE ZIM-DOMAIN) proteins in *Arabidopsis*. Briefly, JAZ proteins are transcriptional repressors that mechanistically link the two previously identified JA signalling components, COI1 (CORONATINE INSENSITIVE1), an F-box protein and a JA-co-receptor that together with SKIP and CULLIN forms the E3 ubiquitin ligase SCF^{Coi1} complex required for specific degradation of repressor proteins, and MYC2, a bHLH transcription factor that regulates diverse JA-dependent genes. When JA (i.e. JA–Ile) levels are low, JAZ proteins acting as repressors of JA signalling interact with MYC2, disrupting both its expression and its transcriptional regulatory activity by the recruitment of co-repressor TOPLESS (TPL) through the EAR (ERF-ASSOCIATED AMPHIPILIC REPRESSION)-domain containing protein NINJA (NOVEL INTERACTOR of JAZ) (Pauwels *et al.*, 2010). When cellular JA levels are elevated as a result of a stress event, binding of JA–Ile to the SCF^{COI1}–JAZ co-receptor complex leads to the degradation of JAZ repressors (Chini *et al.*, 2007; Thines *et al.*, 2007; Sheard *et al.*, 2010). This liberates the transcriptional regulator MYC2 and possibly other transcriptional regulators from suppression, and JA responses are activated. Molecular events involved in JA signalling have recently been extensively reviewed (Staswick, 2008; Chung *et al.*, 2009; Browse *et al.*, 2009; Fonseca *et al.*, 2009; Gfeller *et al.*, 2010; Howe, 2010).

JA–light interplay: major players

Recent genetic and biochemical studies have demonstrated that several components of the JA pathway including the JA co-receptors, COI1 and JAZ proteins, as well as MYC2 and JAR1 influence various aspects of light responses. Similarly, various components of light signalling, including photoreceptor phytochromes, influence JA-regulated gene expression and responses, suggesting a reciprocal interaction between these two signalling pathways. Here, the roles of some of the relatively well-characterized components of light and JA signalling involved in this interplay are briefly reviewed.

COI1

COI1 physically interacts with the COP9 (CONSTITUTIVE PHOTOMORPHOGENIC 9) signalosome (CSN), an evolutionarily conserved multiprotein complex that suppresses photomorphogenesis in the dark through the degradation of the positive regulators, HY5 and HYH transcription factors (Feng *et al.*, 2003). This suggests an interplay between these two signalling pathways at the level of JA reception. Indeed, recent analysis of the *coi1* mutant under different light regimes showed a number of light-associated phenotypes. First, *coi1* flowers earlier under long days than wild-type plants (Robson *et al.*, 2010), a phenotype that is also displayed by the *phyB* mutant. Secondly, *coi1* showed an enhanced SAS response when grown under a low R:FR ratio with hypocotyls 30% longer than those of the wild type, whereas under a high R:FR ratio, *coi1* hypocotyls were not different in length from wild-type hypocotyls (Robson *et al.*, 2010), a phenotype also displayed by the *phyA* mutant. The hypocotyls of *coi1* were also longer than those of the wild type when grown under either cFR or continuous R light, suggesting that COI1 is required for light-mediated inhibition of hypocotyl elongation (Robson *et al.*, 2010).

JAZ repressors and JAR1

Mutants for JAI3/JAZ3 and MYC2 genes that act downstream from COI1 as well as those for the upstream jasmonate biosynthesis genes JAR1 and AOS show enhanced SAS in response to low R:FR light, suggesting that these JA genes are required for hypocotyl growth inhibition by FR light (Robson *et al.*, 2010). Of these, the *jar1* mutant has been independently isolated for its altered response to FR light and named *fin219* (*far-red-insentive219*) (Hsieh *et al.*, 2000).

MYC2

MYC2 appears to act at the cross-roads of JA and various light signalling pathways. *jin1/myc2* mutants show an increased sensitivity to shade or FR light measured as a higher percentage increase in hypocotyl elongation under low R:FR than wild-type plants (Robson *et al.*, 2010). Light-responsive genes were up-regulated by FR and blue light (BL) in the *jin1/myc2* mutant background (Yadav *et al.*, 2005). The *jin1/myc2* mutant also shows enhanced inhibition of hypocotyl elongation under BL, suggesting that MYC2 is a negative regulator of BL-mediated photomorphogenic growth (Yadav *et al.*, 2005). Furthermore, MYC2 binds to the G-box sequence (Gangappa *et al.*, 2010) found in the promoter of *SPA1* (suppressor of PHYA). *SPA1* encodes a negative regulator of photomorphogenesis required for COP1-mediated degradation of HY5 and HFR1 (Saijo *et al.*, 2003). It was deduced from the analysis of the *myc2 spa1* double mutant that MYC2 and SPA1 act redundantly in the dark and synergistically in the light to suppress photomorphogenesis (Gangappa *et al.*, 2010). Furthermore, an antagonistic effect of the *spa1* mutation

on *myc2*-mediated JA responses was observed. In the *myc2* mutant, JA-induced expression of the insect defence gene *VSP2* is reduced while that of the pathogen defence gene *CHIB* is increased relative to the expression of these genes in wild-type plants. Therefore, MYC2 acts as a positive and negative regulator of JA-responsive insect and pathogen defences, respectively (Anderson *et al.*, 2004; Lorenzo *et al.*, 2004). In contrast, in the *myc2 spa1* double mutant, both *VSP2* and *CHIB* are induced by levels of JA similar to those observed in wild-type plants (Gangappa *et al.*, 2010). This result is consistent with the view that MYC2 regulates several nodes whereby JA and light signalling interact. Recently several MYC2-related bHLH transcription factors, such as MYC3 and MYC4, have also been shown to be involved in JA signalling (Cheng *et al.*, 2011; Fernández-Calvo *et al.*, 2011; Niu *et al.*, 2011). However, potential involvement of these transcription factors in light–JA interplay is currently unknown.

PHYA

Further supporting the link between FR light and JA signalling is the observation of reduced sensitivity of root growth inhibition by MeJA in the *phyA* mutant, which also has reduced MeJA induction of the *VSP1* transcript (Robson *et al.*, 2010), a commonly used marker gene for the JA-regulated wound response. Other JA- and COI1-dependent responses such as wound- or JA-mediated growth reduction of leaves and anthocyanin accumulation patterns were not different in the *phyA* mutant, suggesting that PHYA is required for only a subset of JA responses or its effect on JA responses is tissue specific (Robson *et al.*, 2010). Nevertheless, these findings, together with other examples discussed below, suggest a reciprocal interaction between JA and FR light signalling. In this interaction, JA biosynthesis and signalling are involved in modulating plant responses to FR light, while components of FR sensing (e.g. PHYA) are required for correct expression of a subset of JA responses.

Robson *et al.* (2010) also obtained evidence to explain why PHYA differentially affects root- and leaf-specific JA responses. It appears that the PHYA-mediated regulation of JA responses occurs at least partly through JAZ1. The JAZ1 repressor protein is degraded by the 26S proteasome in JA-treated wild-type plants in a COI1-dependent manner as the *coi1* mutant is deficient in JA-mediated repressor degradation (Chini *et al.*, 2007; Thines *et al.*, 2007). Similarly to the *coi1* mutant, JA-mediated degradation of JAZ1 did not occur in the leaves of the *phyA* mutant upon wounding or after MeJA treatment, but did occur in the roots treated similarly (Robson *et al.*, 2010). So, it appears that light perception through PHYA is required for specific activation of JA responses in foliar tissues through the regulation of JAZ protein stability.

PHYB

The interaction of PHYB function with JA signalling is reviewed later in the section on light–JA interplay during SAS.

HY5, HY1, HY2, COP1, and COP9

HY5 encodes a basic leucine-zipper (bZIP) transcription factor that positively regulates photomorphogenesis. HY5 is also proposed to be the major integrator of light and multiple hormone signalling pathways, including JA (Lau and Deng, 2010). Chromatin immunoprecipitation analysis demonstrated that HY5 binds to the promoter of the *LOX3* gene (Lee *et al.*, 2007), which was implicated to be involved in JA biosynthesis (Caldelari *et al.*, 2011), suggesting a possibility that HY5 is involved in regulating JA signal production. HY1 and HY2 are involved in phytochrome chromophore biosynthesis, and *Arabidopsis* mutants, *hy1* and *hy2*, deficient in phytochrome chromophore biosynthesis displayed a JA overproduction phenotype and constitutive activation of the JA-inducible and SCF^{COI1}-dependent genes (Zhai *et al.*, 2007). Aberrant expression of defence genes including JA-dependent defence genes was observed in several light mutants such as *cop1*, *cop9*, and *det1* (Mayer *et al.*, 1996). Finally, the COP9 signalosome is also linked to JA biosynthesis and JA-dependent defences. Silencing of genes encoding CSN subunits in tomato leads to the compromised expression of JA-responsive genes following mechanical wounding and insect attack. Furthermore, CSN-silenced tomato plants show reduced resistance to the necrotrophic pathogen *Botrytis cinerea* as well as to the larvae of the herbivorous insect *Manduca sexta* (Hind *et al.*, 2011). In addition, Geminiviruses (plant DNA viruses) target CSN to inhibit JA biosynthesis required for Geminivirus resistance in *Arabidopsis* (Lozano-Durán *et al.*, 2011). Together, these examples reiterate the view that phytochrome function and light signalling are required for correct expression of JA-dependent responses.

In the following sections, other emerging links between light and JA perception and signalling that influence agriculturally important plant features such as SAS and insect and pathogen defence will be briefly reviewed.

Light–JA interplay during shade avoidance syndrome

As briefly discussed above, the recent demonstration that JA signalling mutants such as *jar1*, *coi1*, *jaz3*, and *jin1/myc2* show exaggerated shade responses under low R:FR conditions where PHYA might antagonize cFR-mediated shade responses (Robson *et al.*, 2010) implicated the JA signalling pathway as a regulator of shade responses. Shady conditions and the SAS present new challenges for plant survival. Drastic alteration of plant morphology associated with elongation (i.e. extended cells and thin cell walls) during SAS could weaken the plant's physical defences. In addition, under shade conditions, pest and pathogen populations can rapidly increase due to increased moisture levels at the lower canopy. Therefore, growth in response to the shade may make the plant vulnerable to pests and pathogens. Indeed, the cucumber mutant, *lhs* (*long-hypocotyl*), lacking a PHYB-like polypeptide and constitutively expressing SAS, sustained

93% more herbivory than its near-isogenic wild-type line (McGuire and Agrawal, 2005). In tomato plants exposed to reflected FR, the performance of the specialist herbivore *M. sexta* (tobacco bollworm) increased and on average caterpillars feeding on the FR-treated plants had 48% more mass than the control plants. Moreover, the *phyB1 phyB2* double mutant showed increased herbivory and insect growth as compared with wild-type plants (Izaguirre *et al.*, 2006). Moreno *et al.* (2009) showed that the *Spodeptera frugiperda* larvae (caterpillars) feeding on *Arabidopsis* plants grown in a crowded arrangement gained significantly more weight than on plants grown in an open canopy. Caterpillar growth on the *phyB* mutant that showed constitutively active SAS was also higher than that on the wild type in both low and high plant densities. The role of light perception on these effects on plant defence was demonstrated by FR light treatment mimicking the effect of plant density on caterpillar growth. It is therefore concluded that SAS, regardless of whether it is induced by crowding or by FR light, makes *Arabidopsis* plants more susceptible to herbivory by insect pests (Moreno *et al.*, 2009).

How does FR light make the plants more susceptible to herbivory? Izaguirre *et al.* (2006) found that FR light caused a dramatic down-regulation of the expression of several defence-related genes, including JA-dependent defence, and inhibited the accumulation of herbivore-induced phenolic compounds. As stated above, jasmonates function in plant defence against pests and pathogens and, in an attempt to find the mechanism of FR-induced susceptibility to insect pests, Moreno *et al.* (2009) examined the regulation of JA-dependent defences by FR light in both the wild type and *phyB* mutants. As suspected, wild-type plants treated with JA under FR light showed reduced induction of *ERF1* (*ETHYLENE RESPONSE FACTOR1*) encoding a JA-responsive AP2/ERF-domain transcription factor as well as the ERF1-regulated pathogen defence genes, *PDF1.2* and *HEL* (Moreno *et al.*, 2009). *ERF1*, *PDF1.2*, and *HEL* are normally associated with defence against pathogens not against herbivores. Therefore, it is not clear how reduced expression of these pathogen defence genes makes the plants more vulnerable to herbivory in FR light-exposed plants. However, FR light-exposed plants were not able to produce leaf phenolics associated with insect defence upon MeJA treatment. Additionally, *phyB* mutants grown under ambient light had lower levels of these phenolics and were not able to produce phenolics when treated with methyl jasmonate (MeJA; Moreno *et al.*, 2009). Therefore, this latter aspect of the JA-dependent defence (e.g. reduced levels of phenolics) rather than reduced fungal defence gene expression might be responsible for making the FR-exposed plants susceptible to certain species of insect pests.

Light–JA interplay during indirect defence against herbivory

In addition to the regulation of defences that are directly effective against herbivores, JA modulates indirect defences that protect the plants from herbivory by recruiting natural enemies of insects. One form of such indirect defence employed by lima bean (*Phaseolus lunatus*) is the secretion of extrafloral nectar (EFN) that is thought to attract insect pollinators (Kost and Heil, 2008; Radhika *et al.*, 2010*a*, and references therein). EFN production also recruits ants, natural enemies of herbivores that feed on lima bean. Importantly, EFN biosynthesis is activated by JA in a light-dependent manner (Radhika *et al.*, 2010*b*). In the dark, exogenous JA inhibited EFN production, whereas in the light, JA activated EFN biosynthesis. In addition, in FR light-exposed plants, JA-mediated EFN production was significantly lower than FR light-unexposed plants, and increasing R:FR ratios restored the EFN secretion rates by JA (Radhika *et al.*, 2010*b*). This result is consistent with the view that FR light negatively influences both direct and indirect defences regulated by JA (see also below).

Do FR light and shade differentially affect different JA-dependent defence responses?

A recent study by Robson *et al.* (2010) has examined basal expression levels of JA-responsive genes in wild-type and *coi1* plants exposed to cFR light in the absence of JA. In contrast to Moreno *et al.* (2009), Robson *et al.* (2010) found that FR light transcriptionally activated the expression of JA biosynthesis (e.g. *AOC1*), signalling (*JAZ1* and *MYC2*), and wound response (*VSP1*) genes. The FR light-induced expression of these genes was attenuated in the *coi1* mutant background, suggesting that FR light is a positive regulator of JA-responsive gene expression. Although this study by Robson *et al.* (2010) may at first appear to be somewhat contradictory to that by Moreno *et al.* (2009), a closer examination of these two studies suggests that FR light differentially regulates different branches of the JA signalling pathway. In fact, MeJA-responsive expression of *MYC2* and *VSP1* in plants treated with FR light was slightly induced, while that of *ERF1* and *PDF1.2* was repressed in the earlier study by Moreno *et al.* (2009). Therefore, it appears that FR light/SAS negatively regulates JA-dependent pathogen defence genes while positively regulating (or priming) JA-dependent wound/insect defence genes, and this may be achieved through differential regulation of *ERF1* and *MYC2*, two key transcriptional regulators of the JA pathway. As depicted in Fig. 1, ERF1 is a positive and negative regulator of JA-responsive pathogen and insect defence genes, respectively. MYC2 has an opposite function to ERF1 in that MYC2 negatively and positively regulates pathogen and insect defence genes, respectively (Lorenzo *et al.*, 2004; Dombrecht *et al.*, 2007).

In another study, lateral shading was found to enhance the expression of a different subset of JA-inducible defences in *Arabidopsis* (Cipollini, 2005). Total peroxidase activity was found to be inducible by JA treatment in shaded plants but not in JA-treated unshaded plants. Another insect defence response investigated in laterally shaded plants was the level of trypsin inhibitors known to be inducible by JA

(Cipollini, 2004). Interestingly, shaded plants had increased trypsin inhibitor levels in their leaves relative to unshaded plants in the absence of JA treatment, which equally induced trypsin inhibitor levels in both shaded and unshaded plants (Cipollini, 2005). Although this latter study has not examined whether shade would make the plants more or less susceptible to herbivory, these results are consistent with the view that FR light/SAS differentially affects different JA-dependent defences.

SAS, FR light, and JA-mediated fungal defence

FR light-mediated attenuation of the JA-responsive pathogen defence genes, *ERF1* and *PDF1.2*, in wild-type plants and the reduced transcript levels of these genes in *phyB* mutants (Moreno *et al.*, 2009) suggest that SAS can make the plants more susceptible to fungal pathogens that are sensitive to JA-dependent defences. However, the effect of SAS on pathogen response has not been studied in great detail. The fungal disease resistance of the *phyB* mutant was tested and it was found that this mutant was indeed more susceptible to the fungal pathogen *Fusarium oxysporum* than wild-type plants (Fig. 2A). This, taken together with previous observations of increased herbivore susceptibility of the *phyB* mutant, suggests that the constitutive SAS response operating in the *phyB* mutant could make this mutant more vulnerable to biotic stresses. *Arabidopsis* mutants, *gai1* (see below and Fig. 2B), *jin1*/*myc2* (Anderson *et al.*, 2004), and *pft1* (Kidd *et al.*, 2009), were all compromised in SAS/FR light responses, and JA-dependent defences also show altered resistance to this pathogen.

Defence–competition trade-off

Further research should reveal additional links and complexities between FR light/SAS and JA signalling. However, based on current evidence, it is proposed that weakened JA-dependent insect defences in FR light- or shade-exposed plants could simply be a resource allocation issue. It makes sense that plants that need to deal simultaneously with both pests and pathogens and intruding neighbours must have evolved to make a decision between two alternatives: either to grow, overcome the competition, and reproduce; or to defend by allocating more resources to defence under limited resources (Howe and Jander, 2008; Ballaré, 2009, 2011). It is logical that the latter option might be preferred in the absence of competition but, in the presence of competition, failure to produce offspring would significantly jeopardize the long-term survival chances of a species in competitive environments.

It should be noted, however, that in some cases insect tolerance phenotypes found in shade avoidance mutants such as *lhs* did not correlate with those found in wild-type plants experimentally exposed to neighbour shading (McGuire and Agrawal, 2005). Also, in contrast to shade-intolerant *Arabidopsis* where the defence–SAS trade-off hypothesis is supported, many plant species can tolerate shade and/or have evolved under both intense competition from neighbours and threat by pests and pathogens and thereby can respond equally to both threats. Supporting this view, a recent meta-analysis predicted that the competition–defence trade-offs in diverse plant species may be less common than is often thought (Viola *et al.*, 2010). Also, diverting resources from growth to anti-herbivore defences, only when herbivores are present (i.e. inducible defence), seems to be common in plants adapted to temperate climates. In contrast, plants grown in tropical climates where an ample supply of water and nutrient is present in soil together with constant herbivore presence can both constitutively express anti-herbivore defences and invest in competition strategies (Bixenmann *et al.*, 2010).

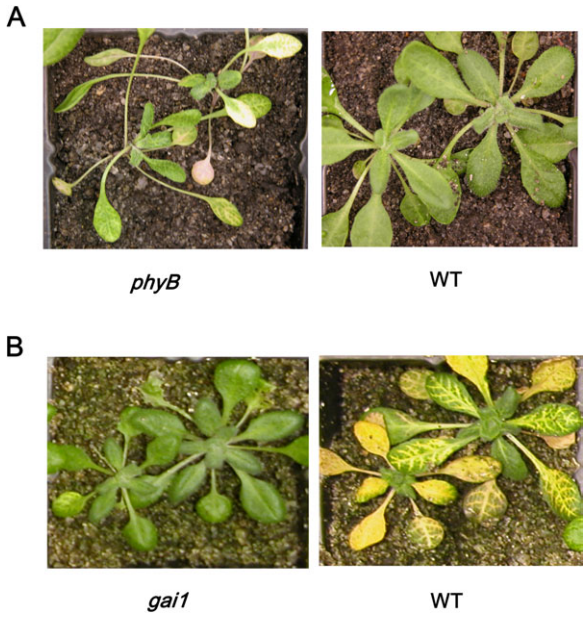

Fig. 2. Shade avoidance syndrome (SAS) and JA-mediated fungal defence. The phytochrome mutant *phyB* (A) constitutively expressing SAS and the DELLA gain-of-function mutant *gai1* (*gibberellin insensitive-1*) (B) show increased and reduced susceptibility, respectively, to the fungal pathogen *Fusarium oxysporum*. Mutants and their corresponding wild-type (WT-*Ler*) plants were inoculated with *F. oxysporum* by dipping the roots of rosette-stage plants into an inoculum of 10^6 spores ml^{-1} as described in Kidd *et al.* (2009). Disease development manifested by veinal clearings and chlorosis of leaves was observed 8 d after inoculations.

Integration of light–JA signalling through DELLA proteins

Recent research has revealed another point of interplay between SAS and JA signalling through growth repressor DELLA proteins. Similarly to PHYB, DELLA proteins inhibit SAS by interacting with PIFs and inhibiting their function (Djakovic-Petrovic *et al.*, 2007). Light induces GA biosynthesis, and GA-mediated degradation of DELLAs

relieves PIF inhibition and promotes SAS (Feng *et al.*, 2008; de Lucas *et al.*, 2008). DELLA proteins, similarly to PHYB, promote JA-responsive defence gene expression under pathogen challenge. The GA-insensitive *gai1* mutant, which has a stabilized DELLA protein resistant to GA-mediated degradation due to a mutation, showed increased JA-responsive gene expression and resistance to the necrotrophic pathogens *Alternaria brassicicola*, *Botrytis cinerea* (Navarro *et al.*, 2008), and *F. oxysporum* (Fig. 2B). In the *coi1 gai1* double mutant, disease resistance observed in the *gai1* mutant against *A. brassicicola* and *B. cinerea*, the two pathogens that are sensitive to JA-dependent defences, was attenuated (Navarro *et al.*, 2008), further supporting the view that the increased pathogen resistance observed in the *gai1* mutant is mediated by JA signalling. Similarly, in a quadruple *DELLA* mutant that contains loss-of-function mutations in four related DELLA proteins, JA-dependent gene defence expression was attenuated. Furthermore, the quadruple *DELLA* mutant showed increased susceptibility to the necrotrophic pathogen *A. brassicicola* (Navarro *et al.*, 2008). These mutant phenotypes further suggest that light signals are integrated into multiple, cross-communicating hormone signalling networks that affect a number of plant traits, including SAS and JA-dependent defence against fungal pathogens.

Integration of light and JA signals through the Mediator complex

The PHYTOCHROME AND FLOWERING TIME1 (PFT1) gene of *Arabidopsis* was proposed to be a positive regulator of PHYB-mediated SAS as *pft1* mutants showed increased and decreased hypocotyl length in FR and R light, respectively, and the *pft1* mutation suppressed the early flowering phenotype of the *phyB* mutant in both short and long days (Cerdán and Chory, 2003). However, under continuous low R:FR conditions, *pft1* was found not to affect flowering time, suggesting that PFT1 may function as a negative regulator of phytochorome signalling as opposed to being a positive regulator of flowering time during shade (Wollenberg *et al.*, 2008). PFT1 has also been implicated in negative regulation of FR light signalling (Wollenberg *et al.*, 2008).

Recent research has shown that PFT1 is an important regulator of JA signalling in *Arabidopsis*. The *pft1* mutant showed reduced levels of JA-responsive gene expression and increased susceptibility to the necrotrophic pathogens *A. brassicicola* and *B. cinerea* (Kidd *et al.*, 2009, 2010). PFT1 overexpression positively regulates JA-responsive defence gene expression and accelerates flowering (Cerdán and Chory, 2003; Kidd *et al.*, 2009). Similarly to *jin1/myc2* and *coi1* mutants, which both showed reduced light- and JA-induced anthocyanin accumulation, the *pft1* mutant displayed reduced expression of the phenylpropanoid biosynthesis gene *PAL* and reduced anthocyanin levels when grown under relatively high light intensities (Fig. 3), further suggesting that PFT1 affects overlapping responses to JA and light. PFT1 encodes the conserved MED25 subunit of the Mediator complex that contains ~30 subunits (Bäckström *et al.*, 2007). The Mediator complex, by coupling the gap between DNA-bound activators and RNA polymerase II, acts as a signal processing centre during transcription (Malik and Roeder, 2010). Therefore, PFT1/MED25 might be required for transmitting the information from transcriptional regulators such as MYC2 and ERF1 to the RNA polymerase II transcriptional apparatus to modulate both basal and JA-responsive expression of fungal defence genes such as *PDF1.2* (Kidd *et al.*, 2009, 2010). The finding that both JA and light signalling require PFT1/MED25 indicates another point of interaction at the level of transcription initiation between light and JA signalling and also is consistent with the conserved function of the Mediator complex as an integrative hub for transcriptional regulation in all eukaryotes (Malik and Roeder, 2010).

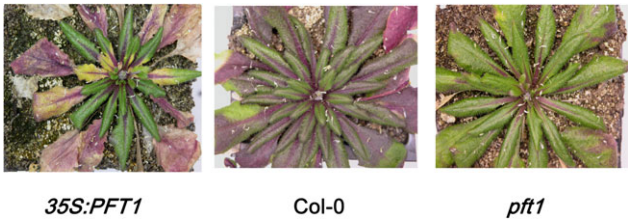

Fig. 3. Light and JA synergistically activate the biosynthesis of stress-related defensive compounds such as anthocyanins. The *pft1* mutant that shows reduced JA-dependent responses also has reduced levels of light-induced anthocyanins in its leaves. In contrast, wild-type plants or plants overexpressing *PFT1* (*35S:PFT1*) show increased anthocyanin production in response to light.

Integration of light and JA signals through chromatin modification

For transcription of eukaryotic genes embedded within chromatin, the recruitment of histone modification enzymes is required. Histone deacetylation is involved in activating transcription while histone acetylation in repressing transcription by reducing the accessibility of the transcription apparatus to promoters (Kouzarides, 2007). Therefore, genes involved in chromatin modifications can potentially integrate signals from multiple pathways. Indeed, recent evidence has shown that at least two histone deacetylase-encoding genes are regulators of both light and JA signalling in *Arabidopsis*. In particular, histone deacetylase RPD3a/HDA19 (also known as HD1) is required for repression of photomorphogenesis as *hda19* mutants show shorter hypocotyls and increased expression of light-inducible genes (*CAB2* and *RBCS1-A*) when grown under FR light (Benhamed *et al.*, 2006). In contrast, transgenic plants constitutively expressing *HDA19* showed increased expression of *ERF1* and ERF1-regulated defence genes as well as increased resistance to the leaf-infecting

necrotrophic pathogen *A. brassicicola*, while *HDA19-RNAi* (RNA inteference) plants had lower levels of JA-responsive genes (Zhou *et al.*, 2005). Collectively, these results suggest that HDA19 antagonistically regulates light and JA responses. Similarly, another histone deacetylase, HDA6, is required for JA-responsive expression of *ERF1*, *PDF1.2*, *MYC2*, and *VSP2* (Wu *et al.*, 2008). COI1 interacts with HDA6 (Devoto *et al.*, 2002), further supporting the role of chromatin modifications in JA-dependent responses. Curiously, as discussed by Memelink (2009), it is not clear why loss of function in histone deacetylases, which are associated with activating transcription, leads to the activation of JA-dependent gene expression.

UV light–JA interplay

The plant receptor for UV-B is not yet known, but particular UV-B treatments induced gene expression patterns that overlap with patterns observed following JA treatment or pathogen attack, suggesting that JA signalling mediates at least some of the UV-B-mediated plant responses. In fact, exposure to UV-B stimulates transcriptional activation of JA biosynthesis genes and rapid JA production (Izaguirre *et al.*, 2003; A.-H.-Mackerness *et al.*, 1999; Schaller, 2001). In *Arabidopsis*, UV-B-mediated expression of stress genes was attenuated in the *jar1* mutant (A.-H.-Mackerness *et al.*, 1999), which shows reduced sensitivity to JA. UV-B, by interacting with JA signalling, also affects the performance of insect pests. For instance, the specialist crucifer insect *Plutella xylostella* L. (diamondback moth) placed more eggs on wild-type *Arabidopsis* plants grown under reduced levels of UV-B light than on plants grown under ambient UV-B and this beneficial effect of UV-B on reduced egg numbers was compromised in the *jar1* mutant (Caputo *et al.*, 2006), suggesting that intact JA biosynthetic and signalling pathways are required for this defensive response.

Similarly in tomato, UV light induces the same set of genes induced by JA and a mutation in the JA pathway blocks this induction (Conconi *et al.*, 1996). An overlap in gene expression induced by either UV-B or systemin, a peptide hormone that activates JA signalling, was also observed in tomato (reviewed by Stratmann *et al.*, 2003). Remarkably, in animals, both UV-B radiation and pathogen infection trigger an inflammatory response in exposed epidermal cells and the synthesis of prostaglandins, which are structurally and functionally similar to jasmonates (Stratmann *et al.*, 2003).

In tobacco (*Nicotiana attenuata*) with a silenced *LOX* gene (*NaLOX3*) and hence impaired JA biosynthesis, UV-B-induced accumulation of phenolic compounds was reduced, suggesting that UV-mediated synthesis of these compounds requires JA (Demkura *et al.*, 2010). In addition, it appears that UV-B primes JA-dependent defences independently from JA levels. The effect of UV-B in priming JA responses contrasts with that of FR light (Demkura *et al.*, 2010), which, as discussed above, down-regulates some specific JA responses.

Excess light–JA interplay

Although light is an essential signal and energy input for growth and development, excess light (EL) has the potential to damage the photosynthetic apparatus. EL is sensed directly by photoreceptors such as phototropin, and cryptochrome (Li *et al.*, 2009). EL also activates both local and systemic light-responsive gene expression which helps the plant to acclimatize to EL-induced stress. This response is known as systemic acquired acclimatization or SAA (reviewed by Li *et al.*, 2009). Recent research has implicated the *Arabidopsis* zinc-finger transcription factor ZAT10 as a modulator of systemic responses to EL (Rossel *et al.*, 2007). The genes that showed alterations in *ZAT10*-overexpressing plants significantly overlap with those altered in JA-treated plants, implicating JA as a possible signal in SAA (Rossel *et al.*, 2007). ZAT10 is induced by 12-oxo-phytodienoic acid (OPDA), an intermediate of JA biosynthesis (Taki *et al.*, 2005). In addition, ZAT10 can bind to the promoter of the JA biosynthesis gene *LOX3* (Pauwels and Goossens, 2008). This finding further implicates ZAT10 in regulating JA biosynthesis as part of a positive feedback loop during exposure to EL.

The capture of light energy in photosynthesis is inefficient and the release of excess electrons creates reactive oxygen species (ROS), and their detoxification by enzymes such as ascorbate peroxidases is a part of the cellular management of photosynthetic activity. It is well established that EL has the potential to produce ROS that oxidize polyunsaturated membrane/plastid lipids such as peroxidation of α-linolenic acid found in plastid membranes. Given that JA biosynthesis is regulated by substrate availability (Wasternack, 2007), it is reasonable to speculate that EL may act to generate JA precursors from chloroplast lipids by non-enzymatic reactions. Indeed, *Arabidopsis* plants lacking PsbS, a ubiquitous pigment-binding protein associated with photosystem II, showed photo-oxidative stress in the chloroplasts as PsbS is involved in non-photochemical quenching required for overcoming potentially detrimental effects of EL. The *psbs* mutants displayed increased expression of genes involved in JA biosynthesis and increased JA levels when subjected to herbivory (Frenkel *et al.*, 2009). It was, therefore, proposed that photo-oxidative stress-mediated transcriptional reprogramming rearranges plant metabolism from growth towards defence that overlaps with that elicited by JA (Frenkel *et al.*, 2009).

The analysis of publically available microarray data (https://www.genevestigator.com/) shows that EL co-ordinately induces the expression of most JA biosynthesis and signalling genes also induced by JA (see also Rossel *et al.*, 2007). However, does light or EL promote JA biosynthesis? An earlier study investigating the effect of light (e.g. dark and 70 μmol and 500 μmol light treatments) on pathogen (*Pseudomonas syringae maculicola* or *Psm*)-induced JA

biosynthesis in *Arabidopsis* has found no effect of light on JA and camalexin levels (Zeier *et al.*, 2004). It was proposed that pathogen-induced salicylic acid (SA) levels may have restricted JA accumulation under light (Zeier *et al.*, 2004), owing to the antagonistic interactions between JA and SA signalling pathways (Kazan and Manners, 2008). However, it is more probable that only EL which would generate ROS that could not be readily removed would have an effect on JA levels.

Recent studies have also indicated a light stress-mediated JA biosynthesis in *Arabidopsis* through the action of a class of proteins called plant fibrillins (Youssef *et al.*, 2010). Fibrillins are structural plastid proteins associated with plastoglobules, which are lipoprotein subcompartments coupled to thylakoid membranes (Austin *et al.*, 2006). A link between JA and EL was proposed based on the finding that phenotypic defects such as retarded shoot growth and the absence of EL-induced anthocyanin production found in plants with reduced expression of genes encoding fibrillin proteins were restorable by exogenous JA application. In addition, expression levels of some JA-responsive genes such as *LOX2* and *VSP2* were reduced in MeJA-treated fibrillin RNAi plants (Youssef *et al.*, 2010). As expected, *Arabidopsis* fibrillin mutants showed altered pathogen resistance (Cooper *et al.*, 2003; Singh *et al.*, 2010), indicating that fibrillins, possibly due to their roles in JA biosynthesis, play a role in plant disease resistance.

The accumulation of anthocyanin pigments is commonly observed in *Arabidopsis* growing under high light intensities and can be exacerbated as the plant ages and defences are weakened (Comparot *et al.*, 2002). Anthocyanin accumulation is controlled by both light and jasmonates, often synergistically (Vázquez-Flota and De Luca, 1998; Curtin *et al.*, 2003; Devoto *et al.*, 2005), among other stress factors. For example, as shown in Fig. 3 for the *pft1* mutant, several light and JA mutants show aberrant regulation of anthocyanin biosynthesis under EL. A link between the light and JA signalling pathways was shown in recent studies that demonstrated that COI1 was essential for JA-induced anthocyanin accumulation (Chen *et al.*, 2007) and this process requires the JA- and light-responsive MYB domain transcription factors PAP1 (MYB75) and PAP2 (MYB90) as well as the bHLH transcription factor GL3 (GLABROUS3) (Shan *et al.*, 2009), the three regulators of phenylpropanoid metabolism genes.

Light effects on JA and JA–Ile synthesis

Although the light dependence of JA biosynthesis was implicated earlier (e.g. Franceschi and Grimes, 1991), more direct evidence about the involvement of light in JA–Ile biosynthesis in lima bean has recently been provided by Radhika *et al.* (2010*b*). These authors found that JA–Ile, which is known to be the biologically active form of JA (Fonseca *et al.*, 2009), rather than JA itself is the signal mediating the production/secretion of the indirect defence molecule EFN in a light-mediated manner. This finding is based on the observation that JA–Ile levels but not JA levels were increased in wounded lima bean leaves exposed to light. In addition, the application of coronal (6-ethyl indanoyl isoleucine conjugate), a structural mimic of JA–Ile, increased EFN secretion rates in the light while inhibitors of the biosynthesis of the amino acid isoleucine reduced EFN secretion rates (Radhika *et al.*, 2010*b*). If light is also required for JA–Ile biosynthesis in *Arabidopsis*, this might explain the reasons behind the failure of earlier studies in finding a link between light levels and JA biosynthesis because only JA but not JA–Ile levels were examined in these previous studies.

As mentioned above, JA regulates wound responses which also seem to be affected by light. A recent study found that the overall wound response of *Arabidopsis* plants was lower in the dark than in the light with respect to both the number and overall expression levels of wound-responsive genes. This effect was associated with a chloroplast-derived signal that appears to originate from the photosynthetic electron transport, and the role of ABA signalling as a potential regulator of this response is ruled out (Morker and Roberts, 2011). Although it is not yet clear whether JA functions as a regulator of this response, it is certainly a strong candidate based on the well-established role of this hormone in wound responses.

JA–light interplay in monocots

The interplay observed between JA and light signalling is by no means restricted to the model plant *Arabidopsis*. Earlier studies have shown that the transcriptional activation of the JA biosynthesis gene *OsAOS1* in rice by R light is activated in a phytochrome-mediated manner (Haga and Iino, 2004). Similarly to *Arabidopsis*, components of JA signalling or biosynthesis affect light sensitivity in rice. For instance, the *Osjar1* rice mutant containing a transposon insertion in the *OsJAR1* gene (also known as *OsGH3-5*) shows increased sensitivity to FR light as *osjar1* coleoptiles (a tissue corresponding to hypocotyl in dicots) were longer under cFR than those of the wild type, suggesting that OsJAR1 behaves similarly to *Arabidopsis* JAR1. In addition, both PHYA and PHYB in rice are required for R light-mediated expression of *OsJAR1*, as the expression of this gene was reduced in individual rice *phyA* and *phyB* mutants and completely abolished in the rice *phyA phyB* double mutant (Riemann *et al.*, 2008).

Another rice mutant called *hebiba* (for snake leaf in Japanese) isolated through a mutant screening shows elongated hypocotyls under saturating R light that normally represses hypocotyl elongation (Riemann *et al.*, 2003). In the dark, however, *hebiba* grows like a wild-type plant. Further experiments showed that R light-mediated activation of the *OsOPR* gene involved in JA synthesis was abolished in *hebiba*. Consistent with this information, *hebiba* contains no or much reduced levels of the JA precursor OPDA and also JA levels. This suggested that the light-associated phenotypes in this mutant were at least partly due to JA deficiency. Indeed, exogenous MeJA

treatment restored these growth defects observed in *hebiba* under R light. Subsequent work showed that light-mediated destruction of PHYA was delayed in *hebiba*, and exogenously supplied MeJA accelerated PHYA destruction in this mutant (Riemann *et al.*, 2009). Finally, comparative analysis of gene expression in *hebiba* versus wild-type rice has led to the identification of the *GER1* (*GDSL CONTAINING ENZYME RICE1*) gene, which encodes a lipase enzyme possibly involved in JA biosynthesis. *GER1* expression was responsive to R and FR light, and to JA (Riemann *et al.*, 2007).

In rice, BL-sensing cryptochromes may be required to promote the light-mediated induction of the JA biosynthesis gene *OsAOS1*. Although no loss-of-function mutants have been characterized for these genes, light-dependent transcription of the putative JA biosynthesis gene, *OsAOS1*, was activated in rice plants transgenically overexpressing the cryptochrome receptor genes, *OsCRY1a* and *OsCRY1b* (Hirose *et al.*, 2006). In maize (*Zea mays*), a novel receptor kinase called WPK1 (WOUND-RESPONSIVE AND PHYTOCHROME-REGULATED KINASE1) was transcriptionally activated rapidly by wounding, JA, and R light, suggesting that WPK1 is involved in JA, wound, and phytochrome signalling. R light also activates the expression of the JA biosynthesis gene *ZmAOS* in maize (He *et al.*, 2005), while the expression of the maize *ZmLOX10* gene responds to the circadian clock with high expression during the daytime (Nemchenko *et al.*, 2006). It should also be noted that JA-responsive genes are significantly represented among circadian clock-regulated genes in *Arabidopsis* (Covington *et al.*, 2008; Mizuno and Yamashino, 2008).

In barley, MeJA treatment reduced aphid numbers when plants were exposed to aphids during natural daylight but not during natural darkness (Glinwood *et al.*, 2007). This observation suggests that light might have an essential role in the differential response of barley to aphids, although the molecular mechanism behind this phenomenon is currently unknown.

The involvement of phytochromes in the regulation of JA-dependent fungal defence also occurs in rice. A recent study showed that the *phyA phyB phyC* triple mutant had lower levels of the JA-responsive defence gene *PR1b* and showed increased susceptibility to the blast fungus *Magnaporthe grisea* (Xie *et al.*, 2011). Together, these findings suggest that JA–light interplay might have an effect on JA-dependent defences in monocots as well.

Hormonal cross-talk affecting light–JA interplay

In this review, the focus has been on the interaction between light and JA perception and signalling. As stated above, light is paramount for many other hormone signalling pathways that directly or indirectly affect JA signalling. PHY and light signalling have often been associated with SA signalling (reviewed by Roden and Ingle, 2009) as SA-induced *PR-1* gene expression is repressed in the wild type in the dark, and in the *phyA*, *phyB*, and *phyA phyB* mutants (Genoud *et al.*, 2002). An antagonistic interaction between SA and JA in *Arabidopsis* is known (reviewed by Kazan and Manners, 2008) and therefore some light-mediated effects attributed to JA could be modulated by SA and vice versa.

Cross-talk between JA and other hormones involved in light effects such as auxin, ethylene, and brassinosteroids have also been reported (Kazan and Manners, 2009; Ren *et al.*, 2009). COI1 is a regulator of ethylene [i.e. 1-aminocyclopropane-1-carboxylic acid (ACC)]-mediated root growth inhibition in the light but not in the dark, as deduced from the analysis of the *coi1* mutant in root growth inhibition tests. However, this effect of the *coi1* mutation was independent from JA biosynthesis and signalling. Neither *aos* nor *opr3* mutants affected in JA biosynthesis nor *jar1* and *jin1*/*myc2* mutants affected in JA signalling showed root growth inhibition by ACC (Adams and Turner, 2010). Therefore, the complex cross-talk among different signalling pathways should be taken into account when examining the role of light mutants on JA responses, and vice versa.

Conclusions

Our understanding of how plants integrate multiple signals is still in its infancy despite significant progress made in this area. Regardless of the mechanisms involved, one thing is becoming evident: complex interplay among different signal transduction pathways, including those regulating defence and development, is a rule rather than an exception. Given that plants have evolved to adapt to diverse light qualities and intensities, it is perhaps not surprising that light perception and signalling intersect with the action of hormones such as jasmonates that affect both development and defence. A better understanding of molecular mechanisms involved in how exogenous and endogenous signals become integrated and processed by plant cells would lead to eventual agricultural benefits for crops subjected to biotic and abiotic challenges.

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Addendum

After acceptance of this paper, Rizzini et al. (2011) have shown that the Arabidopsis UVR8 protein is a UV-B receptor.

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Addendum: After acceptance of this paper, Rizzini *et al*. (2011) have shown that the *Arabidopsis* UVR8 protein is a UV-B receptor (Rizzini L, Favory JJ, Cloix C, Faggionato D, O'Hara A, Kaiserli E, Baumeister R, Schäfer E, Nagy F, Jenkins GI, Ulm R. 2011. Perception of UV-B by the *Arabidopsis* UVR8 protein. Science 332, 103–106.