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REVIEW PAPER

Molecular interaction of jasmonate and phytochrome A signalling

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Abstract

The phytochrome family of red (R) and far-red (FR) light receptors (phyA–phyE in *Arabidopsis*) play important roles throughout plant development and regulate elongation growth during de-etiolation and under light. Phytochromes regulate growth through interaction with the phytohormones gibberellin, auxin, and brassinosteroid. Recently it has been established that jasmonic acid (JA), a phytohormone for stress responses, namely wounding and defence, is also important in inhibition of hypocotyl growth regulated by phyA and phyB. This review focuses on recent advances in our understanding of the molecular basis of the interaction between JA and phytochrome signalling particularly during seedling development in *Arabidopsis*. Significantly, JA biosynthesis genes are induced by phyA. The protein abundance of JAR1/FIN219, an enzyme for the final synthesis step to give JA-Ile, an active form of JA, is also determined by phyA. In addition, JAR1/FIN219 directly interacts with an E3-ligase, COP1, a master regulator for transcription factors regulating hypocotyl growth, suggesting a more direct role in growth regulation. There are a number of points of interaction in the molecular signalling of JA and phytochrome during seedling development in *Arabidopsis*, and we propose a model for how they work together to regulate hypocotyl growth.

Key words: Jasmonic acid (JA), low R:FR light, photomorphogenesis, phytochrome, shade avoidance syndrome, skotomorphogenesis.

Introduction

Global food security has become one of the major concerns in recent years, and understanding plant photomorphogenesis is as important as ever for producing further yield increases for crop species. Photomorphogenesis is a light-regulated developmental programme that controls most aspects of plant development including the regulation of plant architecture (Fankhauser and Chory, 1997). Plants grown in the light typically have short and robust stems with dark-green leaves. In contrast, plants grown in darkness undergo a skotomorphogenic growth strategy and have elongated stems and yellow, etiolated cotyledons or leaves. Plants grown under shade have a morphology that is more akin to that of skotomophogenic growth and have paler leaves with an elongated and weaker stature as they seek to grow taller than their neighbouring plants. This type of response is unfavourable in crop species grown under controlled monocultures in modern agriculture as energy is allocated away from tissues for harvest. For example, >75% of the yield increase in commercial maize hybrids released between 1955 and 2000 is associated with increased tolerance to such competition from neighbours (Duvick, 2005).

Plant responses to shade are not only caused by the reduced level of available light energy, but are primarily due to an enrichment of far-red (FR) light reflecting off neighbouring leaves and stems. Plants have a number of photoreceptors such as UV RESISTANCE LOCUS 8 (UVR8), cryptochromes,

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phototropins, and phytochromes which between them are capable of perceiving light wavelengths ranging from UV-B to FR light (Devlin *et al.*, 2007; Casal, 2013). The blue light photoreceptors, the cryptochromes (Keller *et al.*, 2011) and phototropins (Casal, 2013), are important in perceiving the reduced level of available light energy in shade. The red (R) and FR light receptor phytochromes are the major photoreceptors for plants in perception of the R:FR ratio of light under canopy shade. The plant genome encodes a family of phytochrome genes (i.e. *PHYA–PHYE* in *Arabidopsis*) and the encoded proteins bind to an open tetrapyrrole chromophore, phytochromobilin (Franklin and Quail, 2010). The resulting holoprotein photoreversibly absorbs R and FR light and, to a simple approximation, the R:FR ratio of the light environment is translated to the molecular ratio of R and FR light-absorbing forms of phytochrome. Mutant studies have shown that phytochrome B (phyB) is the primary photoreceptor for R light in growth inhibition regulation of the embryonic stem, the 'hypocotyl', while that for FR light is phytochrome A (phyA). Long hypocotyl mutant screening under monochromatic R and FR light has identified the function of a number of signalling molecules downstream of both phyA and phyB. The molecular basis of phytochrome signalling has been covered comprehensively by a number of excellent reviews (Bae and Choi, 2008; Franklin and Quail, 2010). The plant hormones gibberellin (de Lucas *et al.*, 2008; Feng *et al.*, 2008; Lau and Deng, 2010), auxin (Tao *et al.*, 2008; Halliday *et al.*, 2009; Franklin *et al.*, 2011), and brassinosteroids (Bai *et al.*, 2012; Oh *et al.*, 2012) have all been found to be important in regulating photomorphogenesis (de Lucas and Prat, 2014). In addition, a newer member of the phytohormone family, jasmonic acid (JA), has also been found to be implicated in photomorphogenesis. JAs are oxylipins derived from fatty acids and are important regulators of plant responses to biotic and abiotic stresses, such as defences against pathogen and wounding (Balbi and Devoto, 2008; Rowe and Staswick, 2013; Wasternack and Hause, 2013), and are also involved in pollen maturation and gametogenesis (Wasternack and Hause, 2013). While gibberellins and auxins regulate growth positively, JA serves as a negative regulator in cell elongation (Wasternack *et al.*, 2013). Interaction between JA and other phytohormones has been reviewed in recent years (Lau and Deng, 2010; Kazan and Manners, 2011, 2012; Svyatyna and Riemann, 2012) as has the role of JA in plant defence, response to light, and immunity (Kazan and Manners, 2011; Hua, 2013). phyB has been implicated in the regulation of JA sensitivity in the trade-off in resource allocation between shade avoidance and plant defence (Moreno *et al.*, 2009). Indeed, phyB has the major role in defence regulation under shade at the vegetative stage of *Arabidopsis* development, and this has also been covered by a series of excellent reviews (Ballare, 1999, 2011; Ballare *et al.*, 2012; Cerrudo *et al.*, 2012; de Wit *et al.*, 2013). However, recent studies on the interaction between key regulatory proteins involved in both JA signalling and photo- and skotomorphogenesis have not been covered. The interaction between JA and phyA signalling, in particular, has revealed a number of their important roles in photomorphogenesis. Therefore, in this review, we have focused on the molecular interactions between JA and phyA signalling in photomorphogenesis.

JA in photomorphogenesis

Active JAs in phytochrome-dependent hypocotyl growth regulation

JA is synthesized from α-linoleic acid in the plastid thylakoid membrane (Fig. 1) by lipoxygenases (LOXs), allene oxide synthases (AOSs), and allene oxide cyclases (AOCs), to give 12-oxophytodienoic acid (OPDA). OPDA is then imported into the peroxisome by the ABC transporter, COMATOSE (CTS1) (Theodoulou *et al.*, 2005), is oxidized by oxophytodienoic acid reductase 3 (OPR3), and undergoes three cycles of β-oxidization. The resulting JA is either modified by hydroxylation at the alkyl moiety to permit conjugation with a sugar or sulphate group or, alternatively, is modified at

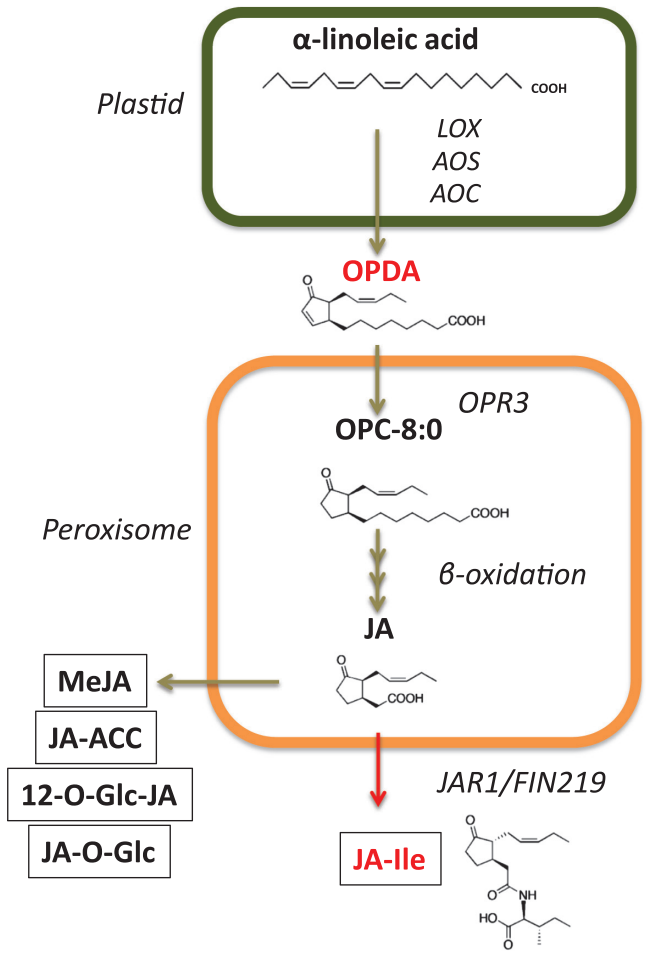

Fig. 1. JA biosynthesis pathway in *Arabidopsis*. JA is synthesized from α-linoleic acid in the chloroplast thylakoid membrane via nuclear genes encoding JA biosynthesis enzymes such as *LOX* (lipoxygenase), *AOS* (allene oxide synthase), and *AOC* (allene oxide cyclase). The JA intermediate 12-oxophytodienoic acid (OPDA) is transported into the peroxisome where it is further processed by β-oxidation. Finally, JA (jasmonic acid) is conjugated with isoleucine by an acyl acid amino synthase, JAR1/FIN219. MeJA, JA-ACC, 12-O-Glc-JA, and JA-O-Glc are all reported to be biologically active.

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the carboxyl moiety with an amino acid or a methyl group to give the biologically active forms (Wasternack *et al.*, 2013). In *Arabidopsis*, conjugation of the amino acid isoleucine to JA to give JA-Ile, an active form of JA, takes place via an acyl acid amino synthetase, JAR1/FIN219, which is specific for JA (Staswick and Tiryaki, 2004). Oxylipin biosynthesis has been proposed to be regulated by negative feedback downstream of JA perception (Stintzi and Browse, 2000), the mechanism of which has yet to be identified.

Phytochrome signalling promotes expression of the genes involved in JA biosynthesis. In rice, the JA biosynthesis genes *AOC*, *AOS1*, and *OsJAR1* are induced by light acting through both phyA and phyB (Svyatyna and Riemann, 2012), and *LOX*, *AOC*, and *JAR1*/*FIN219* genes are up-regulated under FR light by phyA in *Arabidopsis* (Robson *et al.*, 2010). The JAR1/FIN219 protein level is positively regulated by phyA under FR light in a fluence-dependent manner (Wang *et al.*, 2011). It is unlikely that JAR1/FIN219 is the only enzyme for isoleucine conjugation to JA in *Arabidopsis* since the *fin219-null* mutant still contains a small amount of detectable JA-Ile, at least under FR light conditions (Wang *et al.*, 2011). Nevertheless phyA is likely to be the major controller of JA and JA-Ile levels, certainly at the seedling stage in *Arabidopsis*. The rice genome encodes OsJAR1 and OsJAR2. OsJAR1 participates in FR and blue light signalling (Riemann *et al.*, 2008) and has a dominant role in the wounding response of vegetative tissue, while OsJAR2 has overlapping roles with OsJAR1 and both are involved in rice development and defence responses (Wakuta *et al.*, 2011; Shimizu *et al.*, 2013; Svyatyna *et al.*, 2014).

Amongst a number of biologically active forms of JA, JA-Ile binds to and activates an E3-ligase, coronatine insensitive (COI1) (Chini *et al.*, 2007; Thines *et al.*, 2007). Growth of both the root and hypocotyl is repressed by JA, and this is mediated primarily by COI1. The *coi1* mutant along with *phyA*, *jar1*/*fin219*, and *aos* mutants all have significantly longer hypocotyls in FR light compared with the wild type (WT) (Robson *et al.*, 2010), suggesting that JA biosynthesis and the JA-Ile-dependent signalling are critical for phyA-mediated inhibition of hypocotyl elongation. The *coi1* mutant also showed an elongated hypocotyl under R light, suggesting a role also in phyB regulation of hypocotyl growth inhibition (Robson *et al.*, 2010; J. Chen *et al.*, 2013). Interestingly, a JA precursor, OPDA, has also been proposed to inhibit hypocotyl growth (Brux *et al.*, 2008). In this case it has been shown to bind to CYP20-3 and promote the formation of a complex with serine acetyltransferase 1 that functions in stress responses (Park *et al.*, 2013). Whether CYP20-3-dependent OPDA signalling is also important in FR light regulation of hypocotyl growth is yet to be examined. The hypocotyl growth analyses under monochromatic R and FR light using both JA biosynthesis and the JA signalling mutants have provided us with two important pieces of information. One is that COI1 has a role in both R and FR light-dependent hypocotyl growth regulation and, secondly, that there is a signalling pathway independent of COI1 and JA-Ile (J. Chen *et al.*, 2013). JA comes in many forms (Fig. 1), and the biological activity of many of the JA intermediates and derivatives is yet to be verified. To date, the data indicate that phytochrome signalling interacts with JA-Ile and also with other active JA species.

JA and phytochrome signalling are mutually antagonistic

As with any signalling molecule or ligand, JA overload is problematic for plants, and biosynthesis of JA is therefore tightly regulated. There is evidence that a negative feedback loop exists in which JA signalling is inhibited by phytochrome signalling to avoid an overactivation of this signalling pathway. A *phyA* loss-of-function mutant has a significantly higher level of OPDA content compared with that of the WT both in darkness and under FR light (Robson *et al.*, 2010). In addition, the *Arabidopsis* phytochrome chromophore mutants *hy1* and *hy2*, lacking photoactive phytochromes, also have higher levels of JA than the WT and there is constant activation of COI1-dependent JA responses as a result (Zhai *et al.*, 2007). These data indicate that active phytochromes are required for the suppression of JA biosynthesis. It is plausible that phytochrome signalling is playing a part in the proposed negative feedback regulation of oxylipin biosynthesis (Stintzi and Browse, 2000). A number of enzymes have been identified to be involved in JA degradation (Koo and Howe, 2012) and it would be interesting to see if phytochromes regulate the catabolism of JAs. Conversely, JA signalling is important in the regulation of phyA activity. For example, photodestruction of rice phyA is dependent on JA signalling (Riemann *et al.*, 2009), indicating that the turnover of the phytochrome photoreceptor is regulated by JA (Fig. 2A). JA also triggers the phosphorylation of phyA at Ser598 (Ritsema *et al.*, 2010). Although the significance of the latter event has yet to be evaluated, it is possible that JA-dependent stress signalling may directly modulate phyA activity and its degradation. In summary, phytochrome is required for negative feedback regulation of JA biosynthesis, while JA is required for the degradation of phyA. These observations suggest that the two signalling pathways have a mutually antagonistic relationship.

JAZ1 and MYC transcription factors in photomorphogenesis

JA-Ile binding to COI1 activates its E3-ligase activity, targeting a group of transcriptional repressors called JAZ proteins for degradation. JAZs are a family of proteins with a JASMONATE ZIM domain that bind to and repress the activity of MYC transcription factors in the absence of JA (Chini *et al.*, 2007; Thines *et al.*, 2007). JAZ degradation results in activation of the MYC transcription factors for induction of JA and anthocyanin biosynthesis genes and also turns on the downstream JA signal transduction pathway (Fig. 2A). MYC2/JIN1/ZBF1 is a basic helix–loop–helix (bHLH) transcription factor that binds to E-, G-, and Z-box motifs in the promoters of genes regulated by light, abscisic acid (ABA), and/or JA signalling (Yadav *et al.*, 2005; Chini *et al.*, 2007, 2009; Kazan and Manners, 2013). A MYC2 mutant, *jin1-1*, showed an elongated hypocotyl under FR and low R:FR light (Robson *et al.*, 2010), indicating that MYC2 has a positive role in phyA-mediated photomorphogenesis under FR light (Fig. 2B). JAZ1–β-glucuronidase (GUS) overexpressor lines

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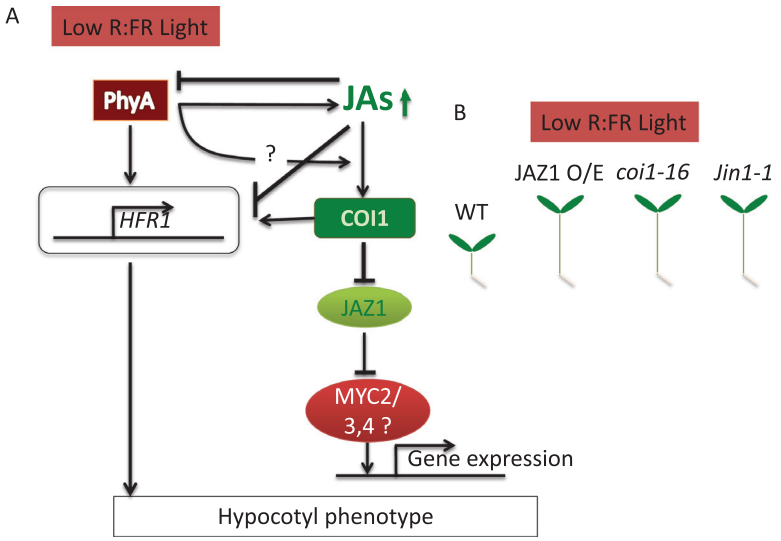

Fig. 2. JA regulation of hypocotyl elongation. (A) Activation of phyA under low R:FR light leads to JA biosynthesis that activates COI1 resulting in degradation of JAZ1. The degradation of JAZ repressors results in the activation of MYC transcription factors that bind to the Z-box in the promoter of genes such as *LHCB* and *SPA1*. PhyA also independently activates the growth regulator HFR1, expression of which is suppressed by JA. (B) *coi1-16* and *jin1-1* mutants and the JAZ1 overexpressor (JAZ1 O/E) show an elongated hypocotyl phenotype under low R:FR light. There are at least two close relatives of MYC2, MYC3 and MYC4, that may also be involved in hypocotyl growth regulation.

show an elongated hypocotyl compared with the WT under low R:FR light (Fig. 2B; Robson *et al.*, 2010), indicating that JAZ1 is preventing photomorphogenesis (represented by the shortening of the hypocotyl). These results are consistent with the known molecular interaction between JAZs and MYCs in which the overexpression of the former and the loss of activity of the latter resulted in the same elongated hypocotyl phenotype. MYC2 has two close relatives, MYC3 and MYC4, in the *Arabidopsis* genome (Fernandez-Calvo *et al.*, 2011). Given that these latter two transcription factors also interact with a number of JAZ proteins, it would be interesting to investigate whether MYC3 and MYC4 also have a role in photomorphogenesis.

It is worth noting that MYC2 was found to interact with ARD1 (acireductone dioxygenase 1; Table 1), a protein also identified in the heterotrimeric G-protein interactome where it interacts with the Gβ subunit (Klopffleisch *et al.*, 2011). *Arabidopsis* lines overexpressing the G-protein α-subunit are hypersensitive to both light and JA signals (Okamoto *et al.*, 2001, 2009), suggesting that the light signal perceived by phytochrome that leads to JA induction is probably enhanced by heterotrimeric G-proteins. It is possible that this is mediated via an association of G-proteins with MYC2 via ARD1, and such a possibility should be further investigated.

JA levels are regulated by the circadian clock

The JA level is also regulated indirectly by phytochromes via the photoreceptor-entrained circadian clock (Goodspeed *et al.*, 2012). Circadian regulation results in a peak in JA levels at subjective midday in *Arabidopsis*, and this is likely to be mediated by the interaction between the key regulators of JA signalling and a number of flowering/circadian regulators that have been identified as interacting proteins (Table 1). In addition, MYC2 binds to the G-box motif in the promoter of SUPPRESSOR OF PHYTOCHROME A-105 1 (*SPA1*) (Gangappa and Chattopadhyay, 2010; Gangappa *et al.*, 2010). SPA1 is a regulator of circadian rhythms and light signalling that acts with MYC2 in suppressing photomorphogenesis; redundantly in darkness and synergistically in the light (Gangappa and Chattopadhyay, 2010; Gangappa *et al.*, 2010). Recent evidence has shown that TIME FOR COFFEE (TIC), a component of the circadian gating of light responses in the *Arabidopsis* nucleus (Hall *et al.*, 2003; Ding *et al.*, 2007), also interacts with MYC2 (Table 1). As a result, MYC2 protein is targeted by the proteasome (Shin *et al.*, 2012). These results indicate that the circadian accumulation of JA leads to an activation of a master regulator, MYC2, that induces the genes under the regulation of the G-box promoter element such as SPA1, a repressor protein for photomorphogenesis, while MYC2 is eliminated later by circadian gating.

Active phytochromes are essential for JA signalling

We have seen a number of examples of an antagonistic relationship between JA and phyA-dependent photomorphogenesis. We also have two examples indicating that a light signal is an essential element in some JA signalling. The first is that JA- and COI1-dependent degradation of JAZ1–GUS in the aerial part of *Arabidopsis* seedlings requires active phyA (Robson *et al.*, 2010). This indicates that photoactive phyA is essential in COI1-dependent signalling. The second comes from a screen to isolate loss-of-function mutants for JA-dependent root growth inhibition, which identified several alleles of phyB including one with a premature stop codon, indicating that active phyB is required for this JA-mediated root growth inhibition (J. Chen *et al.*, 2013). Although the significance of the latter result is yet to be determined, these data both indicate that active phytochromes, functioning in response to their respective light inputs, are essential for JA signalling.

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Table 1. *Interacting proteins of JA signalling components*

Proteins in JA signalling	Interacting protein	Systematic name	Functional Name	Analysis	References
COI1 (At2g39940)	CIP1	At5g41790	COP1-interacting 1	Y2H	Devoto *et al.* (2002)
	CIP4	At5g37190	COP1-interacting 4	Y2H	Devoto *et al.* (2002)
	HD1	At4g38130	Histone deacetylase 1	Y2H	Devoto *et al.* (2002)
	HDA6	At5g63110	Histone deacetylase 6	Y2H	Devoto *et al.* (2002)
	CUL1	At4g02570	Cullin/auxin resistant 6	Y2H, AC, RC	Devoto *et al.* (2002); Xu *et al.* (2002); Potuschak *et al.* (2003); Ren *et al.* (2005)
	SKP1	At1g75950	S-phase kinase associated protein 1	Y2H, AC, RC	Devoto *et al.* (2002); Xu *et al.* (2002); Potuschak *et al.* (2003); Takahashi *et al.* (2004)
	ASK2	At5g42190	*Arabidopsis* SKP1-like 2	Y2H, AC	Devoto *et al.* (2002); Takahashi *et al.* (2004)
	SK11	At4g34210	SKP1-like 11	Y2H	Takahashi *et al.* (2004)
	SK12	At4g34470	SKP1-like 12	Y2H	Takahashi *et al.* (2004)
	COP8/CSN4	At5g42970	COP9-signalosome 4	AC	Feng *et al.* (2003)
	COP9/CSN8	At4g14110	COP9-signalosome 8	AC	Feng *et al.* (2003)
	FUS11/CSN3	At5g14250	COP9-signalosome 3	AC	Feng *et al.* (2003)
	RBX1	At5g20570	Regulator of Cullins-1	AC	Xu *et al.* (2002)
	JAZ1/TIFY10A	At1g19180	Jasmonate ZIM-domain protein (JAZ) 1	AC, Y2H, Co-IP, RC	Thines *et al.* (2007; Melotto *et al.* (2008); Pauwels *et al.* (2010); Yan *et al.* (2013)
	JAZ2/TIFY 10B	At1g74950	Jasmonate ZIM-domain protein (JAZ) 2	Y2H, RC	Chini *et al.* (2007); Melotto *et al.* (2008); Chung *et al.* (2010)
	JAZ3	At3g17860	Jasmonate ZIM-domain protein (JAZ) 3	RC, Y2H	Chini *et al.* (2007); Melotto *et al.* (2008); Chung *et al.* (2010)
	JAZ 9/TIFY 7	At1g70700	Jasmonate ZIM-domain protein (JAZ) 9	RC, Y2H	Melotto *et al.* (2008); Mosblech *et al.* (2011)
	JAZ10	At5g13220	Jasmonate ZIM-domain protein (JAZ) 10	RC	Chung *et al.* (2010)
MYC2/JIN1/JAI1/ZBF1 (At1g32640)	MYC2	At1g32640	MYC2 transcription factor	AC	Fernandez-Calvo *et al.* (2011)
	MYC3	At5g46760	MYC3 transcription factor	RC, Co-IP	Fernandez-Calvo *et al.* (2011)
	MYC4	At4g17880	MYC4 transcription factor	AC	Fernandez-Calvo *et al.* (2011)
	MYB23	At5g40330	MYB transcription factor23	Genetic	Abe *et al.* (2003)
	MYB2	At2g47190	MYB transcription factor2	Genetic	Abe *et al.* (2003)
	JAZ 1	At1g19180	Jasmonate ZIM-domain protein (JAZ) 1	RC, Y2H	Melotto *et al.* (2008); Fernandez-Calvo *et al.* (2011); Niu *et al.* (2011)
	JAZ 3	At3g17860	Jasmonate ZIM-domain protein (JAZ) 3	RC, Y2H	Chini *et al.* (2007); Thines *et al.* (2007); Fernandez-Calvo *et al.* (2011); Niu *et al.* (2011)
	JAZ 2/TIFY10B	At1g74950	Jasmonate ZIM-domain protein (JAZ) 2	RC, Y2H	Fernandez-Calvo *et al.* (2011)
	JAZ 5	At1g17380	Jasmonate ZIM-domain protein (JAZ) 5	RC, Y2H	Fernandez-Calvo *et al.* (2011)
	JAZ 6	At1g72450	Jasmonate ZIM-domain protein (JAZ) 6	RC, Y2H	Fernandez-Calvo *et al.* (2011)
	JAZ 8	At1g30135	Jasmonate ZIM-domain protein (JAZ) 8	RC, Y2H	Fernandez-Calvo *et al.* (2011)
	JAZ 9/TIFY7	At1g70700	Jasmonate ZIM-domain protein (JAZ) 9	RC, Y2H	Fernandez-Calvo *et al.* (2011)

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Table 1. *Continued*

Proteins in JA signalling	Interacting protein	Systematic name	Functional Name	Analysis	References
	JAZ 10	At5g13220	Jasmonate ZIM-domain protein (JAZ) 10	Y2H	Fernandez-Calvo *et al.* (2011)
	JAZ 11	At3g43440	Jasmonate ZIM-domain protein (JAZ) 11	Co-IP, Y2H	Fernandez-Calvo *et al.* (2011)
	JAZ 12	At5g20900	Jasmonate ZIM-domain protein (JAZ) 12	Co-IP, Y2H	Fernandez-Calvo *et al.* (2011)
	GAI	At1g14920	GA insensitive	Y2H	Hong *et al.* (2012)
	RGA1	At2g01570	Repressor of GA1	AC, Y2H	Hong *et al.* (2012)
	RGL1	At1g66350	RGA-like1	Y2H	Hong *et al.* (2012)
	RGL2	At3g03450	RGA-like2	Y2H	Hong *et al.* (2012)
	RGL3	At5g17490	RGA-like3	Y2H	Hong *et al.* (2012)
	ARD1	At4g14716	Acireductone dioxygenase1	Y2H	Klopffleisch *et al.* (2011)
	AHP5	At1g03430	His-containing phosphotransfer	Y2H	Yamashino *et al.* (2003)
	TIC	At3g22380	Time for coffee	AC, PCA, Y2H	Shin *et al.* (2012)
	PFT1	At1g25540	Phytochrome and flowering time 1	AC, PCA, Y2H	Chen *et al.* (2007); Cevik *et al.* (2012)
JAR1/FIN219 (At2g46370)	COP1	At2g32950	Constitutively photomorphogenic 1	Genetic, PCA	Ang *et al.* (1998); Hsieh *et al.* (2000); Wang *et al.* (2011)
	FIP1 (GSTU20)	At1g28200	FIN219 interacting protein 1	Y2H, RC, AC	Chen *et al.* (2007)
	JAR1/FIN219	At2g46370		Y2H, RC, PCA	Wang *et al.* (2011)
OPR3 (At2g06050)	AGD12	At4g21160	ARF GAP domain 12	Y2H	Braun *et al.* (2011)
	Coilin	At1g13030		Y2H	Braun *et al.* (2011)
LOX2 (At3g45140)	NDL1	At5g56750	N-MYC down regulated-like 1	Y2H	Klopffleisch *et al.* (2011)
	EIF4E	At4g18040	Eukaryotic translation initiation factor 4E	Y2H, Co-IP, AC, RC	Freire *et al.* (2000)
LOX3 (At1g17420)	Copine	At1g67800	Calcium-dependent phospholipid-binding protein	Y2H	Braun *et al.* (2011)
AOS (At5g42650)	HK2	At5g35750	Histidine kinase 2	Y2H	Dortay *et al.* (2008)
VSP1 (At5g24780)	VSP1	At5g24780	Vegetative storage protein 1	Crystal Coex	Chen *et al.* (2012)
	GRF10	At1g22300	General regulatory factor 10	AC-MS	Swatek *et al.*, (2011)
	GRF1	At4g09000	General regulatory factor 1	AC-MS	Swatek *et al.*, (2011)
	AG	At4g18960	MADS-box transcription factor, Agamous	Co-IP	Gamboa *et al.*(2001)
	ATARCA	At1g18080	WD40-repeat protein	PCA	Kundu *et al.* (2013)
ERF1 (At3g23240)	PFT1	At1g25540	Phytochrome and flowering time 1	Y2H, RC, PCA	Ou *et al.* (2011); Cevik *et al.* (2012)
	BPM1		BTB-POZ and MATH domain1	Y2H	L. Chen *et al.* (2013)

AC, affinity capture–western; AC-MS, affinity capture–mass spectrometry; RC, reconstituted complex; Y2H, yeast two-hybrid; Co-IP, co immunoprecipitation; PCA, protein fragment complementation assay.

JA in skotomorphogenesis

E3-ligases, COI1 and COP1, antagonistically regulate skotomorphogenesis

Skotomorphogenesis is maintained by a group of proteins CONSTITUTIVELY PHOTOMORPHOGENIC/DE-ET IOLATED/FUSUCA (COP/DET/FUS) that are conserved in eukaryotes across the phyla. The COP9 signalosome and COP1 (Deng *et al.*, 1991, 1992) are central repressors of phototomorphogenesis (Lau and Deng, 2010) that function to regulate protein degradation. In darkness, photomorphogenesis-promoting transcription factors such as the b-ZIP protein HY5 are targeted by COP1 E3-ligase for degradation in the nucleus, while, in the light, COP1 is excluded from the

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nucleus by the action of the photoreceptors, thereby permitting HY5 proteins to function (Fig. 4). Significantly, many of the COP9 signalosome (CSN) proteins (Feng *et al.*, 2003) and the COP1-interacting proteins, CIP1 and CIP4 (Devoto *et al.*, 2002), have been found to interact directly with COI1 (Table 1). It is not known how these *in vitro* interactions lead to biological function; however, they may potentially have a role in chlorophyll metabolism in darkness. Protochlorophyllide (Pchlide) and the enzyme, protochlorophyllide oxidoreductase A (PORA), that catalyses Pchlide reduction to chlorophyllide, both accumulate in seedlings grown in darkness. A rice JA biosynthesis mutant *hebiba* was found to accumulate excess Pchlide in darkness compared with the WT (Sineshchekov *et al.*, 2004), indicating that JA prevents Pchlide accumulation in darkness. COP1 is required for accumulation of PORA in darkness while COI1 probably suppresses *PORA* gene expression in seedlings grown in darkness (Robson *et al.*, 2010). These results indicate that JA signalling mediated by COI1 is acting antagonistically to COP1-dependent skotomorphogenesis. It will be interesting to determine whether interactions of COI1 with CIP1, CIP4, CSN3, CSN4, or CSN8 are directly responsible for the role of JA signalling in the regulation of skotomorphogenesis.

phyA-mediated suppression of PORA accumulation under FR light results in photobleaching in young seedlings when they are subsequently exposed to white light. This block of the greening response in young seedlings by FR light is associated with elevated unbound Pchilde and reduced PORA levels and can be rescued by PORA overexpression (Sperling *et al.*, 1997). Consistent with the demonstration in darkness that JA can suppress accumulation of PORA, both *coi1* and *fin219* mutants are resistant to FR light block of greening (Robson *et al.*, 2010; Wang *et al.*, 2011), indicating that the block of the greening response by FR light is at least in part regulated by COI1 via JA-Ile. Greening in darkness and in FR light may also be regulated by the direct interaction between JAR1/FIN219 and COP1 (discussed below), and this possibility should be tested.

JAR1/FIN219 in skotomorphogenesis

The far-red insensitive, *fin219* mutant was originally identified as a suppressor of the *cop1* mutant (Ang *et al.*, 1998). The enzyme activity for acyl-adenylate/thioester conjugation was subsequently confirmed by additional mutant alleles showing insensitivity to JA (Staswick and Tiryaki, 2004). The JA-Ile-bound JAR1/FIN219 crystal structure (Westfall *et al.*, 2012) confirmed a critical role for the S101 residue in substrate binding, mutation of which in the *jar1-1* allele (S101F) resulted in a loss of enzyme activity (Fig. 3). Given the enzyme activity of JAR1/FIN219, loss-of-function mutations in this gene would be expected to result in a severe reduction of active JA-Ile, thus abolishing JA responses. However, the story may not be as simple as this.

JAR1/FIN219 has also been found to interact directly with COP1 under continuous FR light and in darkness (Wang *et al.*, 2011), confirming the genetic interaction. COP1 binds to the C-terminal coiled-coil domains of FIN219 shown in the crystal structure in Fig. 3, and this domain is in close vicinity

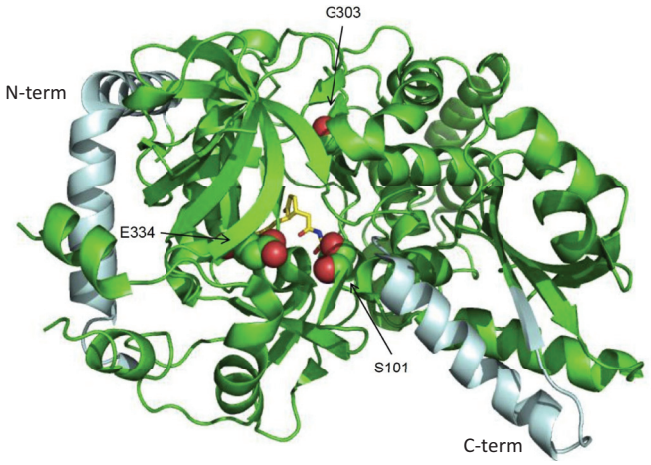

Fig. 3. A ribbon diagram of the crystal structure of JAR1/FIN219 (GH3.11: PDB; 4EPL) with JA. The coiled-coil domains of the JAR1/FIN219 N- (amino acids 9–42) and C-terminus (amino acids 435–461) are indicated in pale blue. The JA molecule is shown with carbon, oxygen, and hydrogen atoms in yellow, red, and dark blue, respectively. Amino acid residues Ser101 (S101) and Glu334 (E334) that were found to be critical for JA-Ile conjugation by analysis of the amino acid substitution alleles of *jar1-1* and *1-3*, respectively, along with Gly303 (G303) that is altered in the *jar1-5* mutation, are indicated with spheres. Carbon and oxygen atoms are shown in green and red, respectively. JAR1/FIN219 interacts with COP1 at the C-terminal coiled-coil domain (pale blue). The N-terminal coiled-coil shown in pale blue has a dominant-negative effect on hypocotyl growth, and overexpression of this domain in *Arabidopsis* results in an elongated hypocotyl phenotype.

to the predicted JA-Ile-binding pocket. The binding of COP1 to JAR1/FIN219 may interfere with JA-Ile binding to the catalytic site. However, the *jar1-1* allele with the S101F mutation in the JA-binding site was more sensitive to monochromatic FR light than the *fin219* allele. It is plausible that in addition to JA and Ile conjugation activity, JAR1/FIN219 has a separate regulatory function. Further analysis of the *jar1-3* (E334K) and *jar1-5* (G303R) alleles (Staswick *et al.*, 2002) in the context of photomorphogenesis may resolve the precise role of JAR1/FIN219 function in JA and photomorphogenic signalling. Photoreceptor-regulated COP1 nuclear exclusion allows HY5 accumulation in the nucleus (Fig. 4) with the consequent inhibition of hypocotyl elongation (Hardtke *et al.*, 2000; Osterlund *et al.*, 2000*a*, *b*). JAR1/FIN219 was found to interact with COP1 in the cytoplasm (Wang *et al.*, 2011) (Fig. 4), and overexpression of full-length JAR1/FIN219 resulted in a hyper-photomorphogenic phenotype. JAR1/FIN219 may enforce exclusion of COP1 from the nucleus and this may lead to an increase in HY5 accumulation in the nucleus resulting in hyper-photomorphogenic development of seedlings. This may place JAR1/FIN219 as a potential regulator for COP1 nuclear exclusion. Such a hypothesis is especially significant as the mechanism regulating the COP1 shuttle between the cytoplasm and the nucleus is yet to be clarified.

JAR1/FIN219 has been co-purified with FIP1, a glutathione *S*-transferase (GST)-binding protein (Chen *et al.*, 2007), and has also been identified amongst the proteins associated with the vacuolar membrane (Staswick *et al.*, 1998). The significance of this is not immediately apparent; however, DET3 encodes the C subunit of the vacuolar H^+-ATPase (V-ATPase), and

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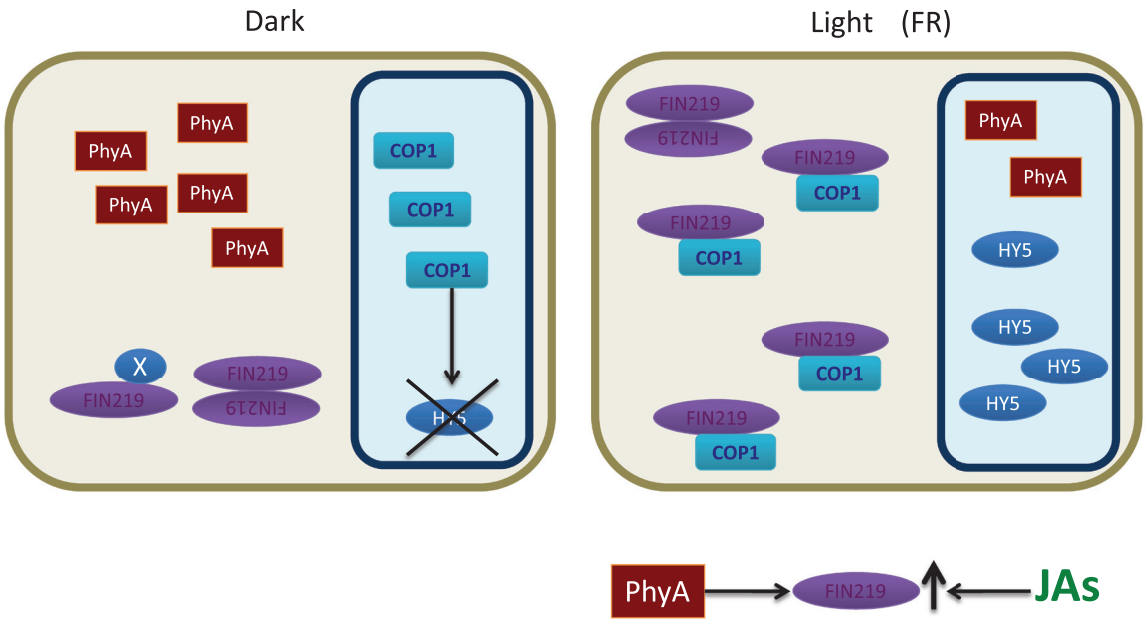

Fig. 4. The role of JAR1/FIN219 in regulation of de-etiolation under FR light. PhyA and JAR1/FIN219 (FIN219) localize in the cytoplasm while an E3 ubiquitin ligase, COP1, is localized in the nucleus where it targets the b-ZIP transcription factor HY5 for degradation in darkness. JAR1/FIN219 proteins are either homodimerized or interact with factor X that is yet to be identified in the cytoplasm. Upon activation by FR, phyA is transported into the nucleus, FIN219 protein starts to accumulate in the cytoplasm, and COP1 shuttles back to the cytoplasm. HY5 protein accumulates as a consequence of nuclear exclusion of COP1 (Osterlund *et al.*, 2000*a*). JAR1/FIN219 protein accumulates in the cytoplasm under FR light. JAR1/FIN219 binds to COP1 in the cytoplasm under FR light (Wang *et al.*, 2011), and this may be a mechanism for the cytoplasmic localization of COP1 under FR.

the *det3-1* mutant was found to overaccumulate the JA precursor, OPDA (Brux *et al.*, 2008). Given the dark de-etiolated (constitutively photomorphogenic) phenotype of the *det3-1* mutant, it is possible that JAR1/FIN219 may also function together with the V-ATPase (Krebs *et al.*, 2010; Schumacher and Krebs, 2010; Okamoto and Futai, 2013), and this is an area that may warrant further study.

Summary

We propose here a current model of hypocotyl regulation by phyA and JAs under FR and low R:FR light (Fig. 2). An activation of phyA by FR light leads to induction of JA biosynthesis. JAs activate COI1 that targets JAZ1 for degradation and allows the MYC2 transcription factor to induce gene expression. Under these conditions, JAR1/FIN219 also regulates COP1 nuclear exclusion, allowing HY5 protein accumulation in the nucleus with the consequent inhibition of hypocotyl elongation (Fig. 4). This model highlights a possible interaction between JA and phytochrome signalling during de-etiolation. In this review, we have also presented two major insights into the molecular interaction of JA and phytochrome signalling: first, that active phytochromes are essential for JA signalling under the respective light conditions in which they function; and, secondly, that JA and phytochrome signalling is mutually exclusive and the negative feedback regulation of JA biosynthesis proposed a decade ago may be regulated by phytochrome signalling. It is also clear that communication between these signalling pathways takes place at many steps in plant development and that these serve as some of the numerous checkpoints to ensure the fine tuning of plant adaptation to a diverse and changing environment.

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