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Javier E. Moreno & Carlos L. Ballaré

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REVIEW ARTICLE

Phytochrome Regulation of Plant Immunity in Vegetation Canopies

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Abstract Plant immunity against pathogens and herbivores is a central determinant of plant fitness in nature and crop yield in agroecosystems. Plant immune responses are orchestrated by two key hormones: jasmonic acid (JA) and salicylic acid (SA). Recent work has demonstrated that for plants of shade-intolerant species, which include the majority of those grown as grain crops, light is a major modulator of defense responses. Light signals that indicate proximity of competitors, such as a low red to far-red (R:FR) ratio, down-regulate the expression of JA- and SA-induced immune responses against pests and pathogens. This down-regulation of defense under low R:FR ratios, which is caused by the photoconversion of the photoreceptor phytochrome B (phyB) to an inactive state, is likely to help the plant to efficiently redirect resources to rapid growth when the competition threat posed by neighboring plants is high. This review is focused on the molecular mechanisms that link phyB with defense signaling. In particular, we discuss novel signaling players that are likely to play a role in the repression of defense responses under low R:FR ratios. A better understanding of the molecular connections between photoreceptors and the hormonal regulation of plant immunity will provide a functional framework to understand the mechanisms used by plants to deal with fundamental resource allocation trade-offs under dynamic conditions of biotic stress.

Keywords R:FR ratio · Immune-suppression · Shade avoidance · Novel players

J. E. Moreno (✉)
Instituto de Agrobiotecnología del Litoral, Consejo Nacional de Investigaciones Científicas y Técnicas, Universidad Nacional del Litoral, CC 242 Ciudad Universitaria, Santa Fe, Argentina
e-mail: javier.moreno@santafe-conicet.gov.ar

C. L. Ballaré (✉)
IFEVA, Consejo Nacional de Investigaciones Científicas y Técnicas, Universidad de Buenos Aires, C1417DSE Buenos Aires, Argentina
e-mail: ballare@ifeva.edu.ar

C. L. Ballaré
IIB-INTECH, Consejo Nacional de Investigaciones Científicas y Técnicas, Universidad Nacional de San Martín, B1650HMP Buenos Aires, Argentina

Introduction

Plant health is an important determinant of plant fitness in natural plant communities and crop yield in agroecosystems. Plant health is not only determined by the abundance of potential consumer organisms (*i.e.*, pathogens and pests), but it is also critically dependent on the ability of the plant to mount efficient immune responses, which in turn can be regulated by a number of abiotic and biotic factors. Current efforts to improve crop yield are largely based on the development of cropping systems that maximize light interception by the crop canopy. Plants of shade-intolerant species, which include the majority of those grown as grain crops, respond to crowding (high crop density) with increased shoot elongation. This response allows the plant to avoid being shaded by competitors and keep its leaves in the upper strata of the canopy. The "shade avoidance syndrome" (SAS) is triggered by the perception, *via* specific photoreceptor proteins, of changes in the light environment brought about by the proximity of other plants (for recent reviews, see Ballaré 2009; Casal 2012; Gommers et al. 2013; Pierik and de Wit 2014).

There is increasing evidence that, in addition to triggering SAS, light spectral changes associated with the proximity of neighboring plants down-regulate the expression of plant immune responses against pests and pathogens (for recent reviews, see Ballaré et al. 2012; Ballaré 2014). This down-regulation is likely to help the plant to efficiently redirect resources to rapid growth when the competition threat posed by the proximity of neighboring plants is high. This review is

focused on our current understanding of the molecular mechanisms used by plants to deal with this resource allocation trade-off between growth and defense. In particular, we discuss novel signaling players that are likely to play a role in the mechanisms that depress defense responses when environmental conditions promote growth and SAS.

The Informative Light-Environment: Detection of Plant Competitors

Sunlight reflected by or transmitted through chlorophyll-containing tissues is enriched in far red radiation (FR, 730 nm), because visible wavelengths, such as blue (B, 450 nm) and red (R, 650 nm) light are absorbed strongly by photosynthetic pigments. Therefore, in plant canopies, low R:FR ratios are associated with shading or a risk of future shading due to the proximity of other plants. Plants can detect and respond to the proximity of other plants by using a variety of photoreceptor proteins. Among them, the phytochromes are responsible for the detection of changes in the R:FR ratio (Ballaré 2009; Casal 2012; Smith 2000). Phytochromes have two inter-convertible forms: Pfr (which is biologically active), and Pr, which is inactive. The Pfr form has its absorption peak in the FR region, whereas the Pr form absorbs maximally in the R region. Therefore, under natural (polychromatic) light conditions, the proportion of phytochrome in the Pfr form (*i.e.*, the Pfr/P ratio) is essentially dictated by the R:FR ratio (Smith 2000). The lower the R:FR ratio (*e.g.*, in very dense canopies), the lower the Pfr concentration. Because Pfr inhibits (elongation) growth, inactivation of phytochrome triggers stem elongation and other components of the SAS (Ballaré et al. 1990; Casal 2012; Pierik and de Wit 2014). The Arabidopsis genome contains a small family of five phytochrome genes: *PHYA*, *B*, *C*, *D*, and *E*. The main R:FR photoreceptor in de-etiolated plants is phyB, as *phyB* mutants show a strong constitutive expression of the SAS phenotype even under full-sunlight, and do not respond to changes in the R:FR ratio with increased elongation (Ballaré 2009). By sensing the R:FR ratio, plants can efficiently detect the proximity of neighboring plants, even before they are actually shaded by future competitors (Ballaré et al. 1990). This early response is thought to increase the plant's competitive ability in crowded stands (reviewed in Ballaré 2009; Smith 2000). At the mechanistic level, SAS responses to low R:FR are triggered when low levels of nuclear phyB Pfr allow the accumulation and increased DNA-binding activity of a series of growth-promoting bHLH transcription factors known as PHYTOCHROME INTERACTING FACTORS (PIFs) (Li et al. 2012; Park et al. 2012). Members of the PIF family promote auxin biosynthesis (Hornitschek et al. 2012; Li et al. 2012). In addition, gibberellin (GA) signaling is stimulated by low Pfr levels (Djakovic-Petrovic et al. 2007; Leone et al. 2014), and GAs promote degradation of a group of PIF-repressing proteins known as DELLAs (de Lucas et al. 2008; Feng et al. 2008). Therefore, low R:FR ratios increase the activity of PIFs, which in turn promote growth by activating auxin biosynthesis genes (Hornitschek et al. 2012; Li et al. 2012), and possibly also by directly activating genes involved in cell wall modification (de Lucas et al. 2008; Sasidharan et al. 2010).

JA- and SA-Inducible Defenses. A General Overview

Plants have evolved a complex defense system that copes with different types of attackers. The hormones jasmonic acid (JA) and salicylic acid (SA) control a broad spectrum of inducible defenses. Biosynthesis of both hormones is activated by biotic stress. Jasmonic acid biosynthesis initiates after perception of stress signals associated with herbivory (herbivore-associated molecular patterns, HAMPS) or cell disruption (such as cell damage caused by necrotrophic pathogens, damage -associated molecular patterns, DAMPs) (Erb et al. 2012; Howe and Jander 2008). Jasmonic acid biosynthesis involves three subcellular compartments: it begins with the release of linolenic acid from the inner chloroplast membrane, continues in the peroxisome with a series of β-oxidations and culminates with the synthesis of the bioactive hormone (JA-Ile, JA conjugated with the amino-acid isoleucine) in the cytosol (Wasternack and Hause 2013). Jasmonic acid plays a central role in the control of plant defense responses to insect herbivores, necrotrophic pathogens, and mechanical damage (Browse 2009; Mithofer and Boland 2012). It also has been shown to modulate reproductive development (McConn and Browse 1996; Xie et al. 1998) and vegetative growth and cell cycle (Pauwels et al. 2008; Yan et al. 2007; Zhang and Turner 2008).

Salicylic acid is also an important hormone in plant defense. Two different pathways are involved in SA biosynthesis: the phenylalanine ammonia-lyase (PAL) pathway and the isochorismate (IC) pathway (Dempsey et al. 2011), involving different subcellular compartments including the chloroplasts and cytosol. Salicylic acid is a key hormone controlling plant defense against viral and bacterial pathogens. The current "zig-zag" model for plant disease resistance suggests different layers of pathogen recognition and disease regulation (Jones and Dangl 2006). Pathogen-associated molecular patterns (PAMPs), which are molecular features associated with microbial cells or activities (such as flagellin, chitin, glycoproteins, and lipopolysaccharides), are recognized by plants by means of extracellular pattern-recognition receptors. Pathogen-associated molecular pattern (PAMP) recognition activates PAMP-triggered immunity (PTI). Some pathogens can overcome this PTI response by using suppressors of PTI (collectively known as effectors). In turn, pathogen-resistant plants have evolved additional defense factors that counteract PTI suppression. Plant cells express R (resistance) proteins which can recognize directly or indirectly a pathogen effector. This specific recognition induces the so-

called effector-triggered immunity (ETI). Both PTI and ETI initiate a series of cellular responses including the production of reactive oxygen species, accumulation of SA, and induction of SA-controlled defense genes. Effector-triggered immunity often leads to elicitation of a controlled cell-death program that confines the pathogen to the infected area. Interestingly, evidence from field and laboratory experiments has established an antagonism between JA- and SA-defense pathways. For example, it has been reported that plants attacked by *Pseudomonas syringae* DC3000, which activates the SA pathway, are more susceptible to the necrotrophic fungus *Alternaria brassicicola* (Spoel et al. 2007). Conversely, insect-damaged plants, which activate the JA pathway, were more susceptible to infections by biotrophic pathogens (see review in Pieterse et al. 2012). This hormonal antagonism has been interpreted as a mechanism that helps the plant to tailor its defense repertoire to the specific nature of the attacker.

JA- and SA- Signaling Modules

JA-Ile is perceived by a co-repressor complex consisting of jasmonate-ZIM proteins (JAZ) and CORONATINE INSENSITIVE 1 (COI1), an F-Box protein that provides target specificity to ubiquitination activity of the signalosome complex (SCF^{COI1}) (Katsir et al. 2008). COI1-JAZ interaction is stabilized by the presence of bioactive JA-Ile in the nucleus (Sheard et al. 2010). JAZ proteins are key molecular players in JA-Ile perception and signaling (Kazan and Manners 2012; Shan et al. 2012). JAZ proteins belong to a family of 12 members in Arabidopsis. JAZ proteins bind to and repress key transcription factors involved in the orchestration of the JA response (Chini et al. 2007; Thines et al. 2007; Yan et al. 2007). Therefore, in a healthy plant, JA responses are repressed by JAZ proteins (Fig. 1a). The mechanism of defense activation is based on ubiquitination and later degradation of JAZ proteins. JAZ proteins share two well-conserved domains: Jas and ZIM. The Jas domain has a dual function providing specificity to the repression of cognate transcription factors, like MYC2 (Chini et al. 2007) and encoding for a functional degron (*i.e.*, a domain required for protein degradation) (Sheard et al. 2010; Withers et al. 2012). The ZIM domain has a conserved TIFY motif that is required both for the interaction with other JAZ proteins (homo- and heterodimerization) (Chini et al. 2009; Chung and Howe 2009) and NINJA recruitment (Pauwels et al. 2010). NINJA functions as a bridge protein between JAZs and the co-repressor protein TOPLESS (Pauwels et al. 2010). This co-repressor complex includes histone deacetylases and probably other DNA modifying enzymes that repress target genes (Zhu et al. 2011). The interaction between JAZs and MYC2, the best studied target transcription factor of JAZ proteins, is controlled by one basic amino-acid residue (R) located in the

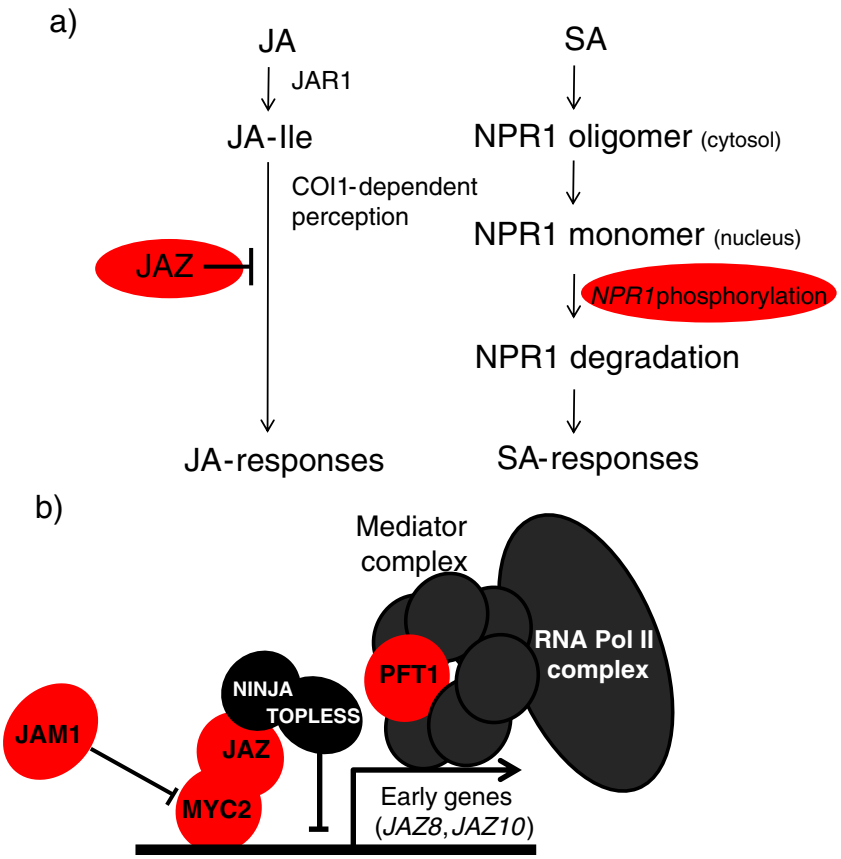

Fig. 1 Light modulation of plant resource allocation to defense responses. **a** Conceptual overview of both jasmonic acid (JA)- and salicylic acid (SA)-signaling pathways, highlighting key events in defense activation. Red ovals indicate some of the signaling events or components that have been shown to be modulated by phytochromes or low R:FR ratio. **b** FR-mediated repression of the JA pathway can result from changes in the expression and stability of key JA-signaling elements. Highlighted in red are those components discussed in the text as possible links between phyB and defense signaling

context of a basic KRK motif at the C-terminal fraction of the Jas domain (Withers et al. 2012). Upon insect damage or a mechanical stress, JA-Ile accumulates in the affected tissues and also in other parts of the plant, triggering the systemic induction of JA-responsive genes (Koo et al. 2009). Transmission of a membrane depolarization signal mediated by GLUTAMATE RECEPTORS-LIKE (GLRs) is thought to be involved in the propagation of the systemic signal to distant tissues, inducing JA biosynthesis and activation of JA-inducible genes (Glauser et al. 2009; Mousavi et al. 2013). The electrical nature of the signaling mechanism may explain the short times required to induce JA-responses in distant tissues, with an estimated signal speed of 7 cm/min in a fully expanded Arabidopsis leaf.

Advances in the understanding of the SA signaling pathway were initially promoted by the characterization of Arabidopsis mutants with a deficient systemic accumulation of PATHOGENESIS RELATED (PR) proteins. This approach has led to the identification of *NONEXPRESSOR of PR* 1 (*NPR1*), which is a central regulator of SA signaling (Cao et al. 1994). A recent breakthrough in the SA-signaling field was the functional characterization of the SA-perception and signaling module. NPR3 and NPR4 were described as SA receptors that regulate NPR1 protein levels in the nucleus (Fig. 1a) (Fu et al. 2012; Wu et al. 2012). Nuclear-localized monomers of NPR1 directly interact with TGA transcription factors to control SA-inducible genes. The current model is still under discussion given conflicting lines of evidence regarding whether or not NPR1 directly binds SA and functions by itself as *bona-fide* receptor for SA (Fu et al. 2012; Wu et al. 2012). A more integrative model suggests that SA could be sensed by a multireceptor complex where different NPR proteins might play a role, depending on specific environmental and developmental conditions (Pajerowska-Mukhtar et al. 2013). NPR1 protein levels and activity are tightly regulated (Mukhtar et al. 2009). In a basal state, oligomers of NPR1 accumulate in the cytosol and, therefore, SA-responses are not activated. Upon pathogen attack and SA accumulation, a more reducing cytosol environment increases the pool of monomeric NPR1. These monomers expose a functional bipartite nuclear localization signal that allows NPR1 traffic to the nucleus and consequently, activation of SA-regulated genes in the presence of TGA transcription factors (Kinkema et al. 2000; Spoel et al. 2009). Nuclear NPR1 stability also is controlled at the post-transcriptional level by phosphorylation (Spoel et al. 2009). This additional layer of regulation of NPR1 stability was shown to be required for full induction of NPR1-controlled genes and a complete development of SA-induced responses (Spoel et al. 2009).

Competition and Defense –A Matter of Balance

In nature, plants are simultaneously exposed to competition with other plants and to the attacks of heterotrophic organisms that feed on plant tissues (herbivores and pathogens). Therefore, plants must be able to grow fast enough to compete with their neighbors and, simultaneously, defend their tissues from pest and pathogens. Plant growth (biomass accumulation) and defense may compete for resources, creating a trade-off in resource allocation that is often referred to as the "dilemma of plants": to grow or defend (Herms and Mattson 1992) (see reviews in Ballaré 2011; Ballaré 2014; Huot et al. 2014; Pierik et al. 2014). Plants allocating resources to rapid growth and expansion of new tissues often display weak defense phenotypes. Traditionally, this reduced investment in defense was interpreted as a direct consequence of diverting resources to rapid growth, at the expense of other physiological functions. Recent studies on the molecular mechanism have revealed that the allocation shifts that plants make in response to changes in the pressure of competition involve sophisticated signaling, and that the photoreceptor phyB plays a fundamental role controlling the expression of hormonal pathways that regulate the plant immune system (reviewed in Ballaré 2014). The following experimental results provide empirical evidence for this general conclusion: 1) plants grown at high density or under natural or simulated shade often display a weak defense phenotype in insect-growth and pathogen infection bioassays (Cerrudo et al. 2012; de Wit et al. 2013; Moreno et al. 2009) (see reviews in Ballaré 2014; Roberts and Paul 2006); 2) Adding FR to the light received by plants without affecting the level of photosynthetically-active radiation was sufficient to reduce plant resistance to a range of insect consumers and plant pathogens (Cerrudo et al. 2012; de Wit et al. 2013; Izaguirre et al. 2006; Moreno et al. 2009); 3) This effect of FR supplementation increasing plant susceptibility to insects (Moreno et al. 2009) and pathogens (Cerrudo et al. 2012) was conserved even in a mutant (*sav3*) (Tao et al. 2008) that fails to elongate in response to low R:FR, thus indicating that the FR effect is not a simple by-product of resource diversion to produce the SAS phenotype; 4) *phyB* mutants suffered more insect herbivory than the corresponding wild-type genotypes in field experiments, and *phyB* tissues supported more insect growth in feeding bioassays (Izaguirre et al. 2006; McGuire and Agrawal 2005; Moreno et al. 2009); 5) constitutive SAS-expressing mutants connected to phyB, *e.g.*, *phyb*, *phya*/*phyb*, and *csa*, were more susceptible to pathogen infection (Cerrudo et al. 2012; Faigón-Soverna et al. 2006; Genoud et al. 2002); 6) FR supplementation desensitized plant tissues against exogenous JA (Izaguirre et al. 2013; Kegge et al. 2013; Leone et al. 2014; Moreno et al. 2009) and SA (de Wit et al. 2013). Taken together, these data indicate that photoconversion of phyB to the inactive Pr form in FR-enriched light environments is sufficient to down-

regulate the key hormonal pathways that control plant immunity, with consequences for plant resistance to pathogens and herbivores (Ballaré 2014).

Light Signals of Competition and Down-Regulation of Plant Immunity: Molecular Mechanisms

JAZ-DELLA Interactions. A Pivotal Point in the Balance?

The mechanisms underlying the effect of R:FR ratio on JA sensitivity are still not well understood, but are likely to involve changes in the levels of JAZ and DELLA proteins. JAZ proteins are emerging as important modulators of multiple plant responses. JAZ proteins can interact with different transcription factors and regulators that, in turn, control several aspects of plant growth and development. These JAZ interactors include DELLAs (Hou et al. 2010; Yang et al. 2012), MYC2/3/4 (Chini et al. 2007; Fernández-Calvo et al. 2011), EIN3 and EIL1 (Zhu et al. 2011), and GL3 and MYB75 (Qi et al. 2011; Song et al. 2011). The conserved GRASS domain of DELLA proteins interacts with several JAZ proteins, including JAZ1, JAZ3, JAZ4, JAZ9, JAZ10, and JAZ11 (Hou et al. 2010; Yang et al. 2012), and this interaction interferes with the ability of JAZs to repress MYC2 and presumably other transcription factors (Hou et al. 2010). This explains why DELLAs are positive regulators of JA signaling (Leone et al. 2014; Navarro et al. 2008; Yang et al. 2012). In turn, DELLA proteins are negative regulators of growth and elongation responses, as they repress the action of growth-promoting PIF transcription factors (de Lucas et al. 2008; Feng et al. 2008). The interaction with JAZ proteins prevents DELLAs from binding to PIFs and repressing growth responses (Yang et al. 2012). DELLA proteins are targeted for degradation by the SCF^{SLY} complex under conditions that promote increased GA synthesis (Hirano et al. 2008). Therefore, the balance between JA and GA hormone levels can affect the relative abundance of JAZ and DELLA proteins, and, consequently, the balance between JAZ-dependent repression of defense responses and DELLA-dependent repression of growth responses (Yang et al. 2012). Low R:FR ratios can result in increased GA signaling and DELLA degradation (Djakovic-Petrovic et al. 2007; Leone et al. 2014), which could represent an important mechanism by which phyB inactivation frees up JAZ proteins to repress JA-responses (Ballaré 2014). In agreement with this hypothesis, it has been recently shown that low R:FR ratios trigger a rapid degradation of DELLA proteins, and that the effects of supplemental FR repressing JA responses are missing in the *gai1* gain-of-function mutant, which encodes a variant of the GAI1 DELLA protein that is resistant to GA-induced degradation (Leone et al. 2014).

JAZ Proteins with Increased Stability: JAZ10.4 and JAZ8

The desensitization of plant tissues toward JA induced by low R:FR ratio could involve increased expression and stability of JAZ proteins (Fig. 1b). JAZ proteins undergo alternative splicing (AS) on the Jas domain, generating Jas-truncated isoforms. AS on the Jas domain can result in the partial or total loss of the COI1-interacting degron. These alternative isoforms show an increased stability due to a weaker interaction with COI1 (Chung et al. 2010). Given that stable isoforms are generated when JAZ genes expression levels are high, it was speculated that JAZ stable isoforms could be part of a negative feedback loop to dampen negative effects of JA on plant cells (Chung and Howe 2009). Within the Arabidopsis JAZ family, JAZ10.4 is the only AS isoform lacking the whole Jas domain, making it resistant to COI1-mediated degradation (Chung and Howe 2009). In contrast to most of the other JAZ proteins whose MYC2-interaction domain relies exclusively on the Jas domain, JAZ10 encodes for a cryptic MYC2 binding domain (CMID) located at the N-terminal part of the protein (Moreno et al. 2013). A functional CMID was required to interact with MYC2 in yeast and pull-down assays (Moreno et al. 2013). Furthermore, JAZ10.4 expression from the *JAZ10* native promoter is able to complement the JA-hypersensitive phenotype of a *jaz10* loss-of-function mutant (*e.g.*, *jaz10-1*) in root length inhibition assays (Demianski et al. 2012; Moreno et al. 2013). Whether FR contributes to increase JAZ10.4 stability or modifies its kinetics of accumulation is still unclear. However, there is genetic evidence suggesting that JAZ10 is involved in the mechanism by which low R:FR ratios repress JA signaling: the effect of FR increasing Arabidopsis sensitivity to *Botrytis cinerea* was significantly attenuated in *jaz10-1* null mutant and in RNA interference lines (Cerrudo et al. 2012). Similarly, the effect of FR repressing JA-induced growth inhibition (Leone et al. 2014) and accumulation of indolic glucosinolates was missing in *jaz10-1* plants (M. Cargnel, P. Demkura and C.Ballaré, unpublished data).

A low R:FR ratio also might enhance expression and stability of JAZ proteins with inherently low turn-over rates. The enhanced stability of JAZ8 has been linked to structural features of its Jas domain (Shyu et al. 2012). Based on the understanding of the structure of the JA-Ile co-receptor complex (Sheard et al. 2010), the increased stability of JAZ8 can be explained by the lack of the LPIAR motif, which reduces the affinity of the hormone for the JAZ8-COI1 receptor complex (Shyu et al. 2012). Recent work, using transgenic Arabidopsis plants constitutively expressing several JAZ-GUS fusion proteins, showed that most of the JAZ proteins, except JAZ2, JAZ6, and JAZ8, accumulate to higher levels in plant growing with white light supplemented with FR radiation than in plants receiving white light alone (Chico et al. 2014). Using a different set of light conditions and Arabidopsis plants constitutively expressing JAZ10- and

JAZ1-GUS fusion proteins, another study showed that supplemental FR radiation could retard JA-induced degradation of JAZ10-GUS, but not JAZ1-GUS (Leone et al. 2014). It remains to be shown if the effect of FR increasing JAZ protein stability is conserved when the JAZ proteins accumulate to endogenous levels, and whether or not the spectrum of JAZ proteins whose stability is affected by phyB manipulations is dependent on the intensity of the R:FR signal.

FR Light Delays the Turnover Rate of bHLH IIIe Transcription Factors

Several basic helix-loop-helix (bHLH) transcription factors from clade III are involved in the regulation of JA-mediated developmental and defense responses. The best known member of this clade is MYC2, a positive regulator of both blue light- and JA-signaling pathways. *MYC2*, *MYC3*, and *MYC4* are phylogenetically related genes (subclade IIIe) that control, in a synergistic way, different aspects of JA-responses (Fernández-Calvo et al. 2011). MYC2/3/4 proteins have an intrinsic high turnover rate regulated by phosphorylation (Zhai et al. 2013). MYC2/3/4 degradation also can be regulated by JA and light. Whereas JA increases the stability of MYC2/3/4 proteins, their degradation is enhanced by supplemental FR radiation through an unknown (COI1-independent) mechanism (Chico et al. 2014). *phyB* mutant plants showed reduced intrinsic levels of MYC2/3/4 protein accumulation suggesting that the active form of phyB is required for MYC2/3/4 stability *in vivo*, and plants of the *myc2/3/4* triple mutant were highly susceptible to *Botrytis cinerea* both under low and high R:FR ratio (Chico et al. 2014). These results are consistent with the idea that at least part of the effect of low R:FR ratios reducing plant sensitivity to JA is mediated by an enhanced turnover rate of MYC proteins. This is unlikely to be the only mechanism, because effects of phyB manipulations on JA sensitivity have been reported using marker genes that are not thought to be regulated by MYCs (such as *ERF1* and *PDF1.2*) (Cerrudo et al. 2012; de Wit et al. 2013; Moreno et al. 2009) . Conceivably, the effect of low R:FR ratios repressing JA responses could result from a combination of mechanisms, where the outcome (strength and breadth of the repression) may depend on the cellular levels of the bioactive JA and GA, *JAZ*/*MYC* gene expression levels, turnover rates of specific JAZ, DELLA, MYC, and PIF proteins, and the strength of the negative feedback loop exerted by stable JAZ isoforms.

bHLH Subclade IIId: The JAM1 Case

Recent work has found that members of subclade IIId of bHLH protein family are negative regulators of the JA signaling pathway (Fig. 1b) (Nakata et al. 2013). The first of these transcription factors was named JA-ASSOCIATED MYC2-LIKE1 (JAM1, bHLH13) due to sequence similarity to MYC2. Despite the fact that bHLH proteins are known to homo- and hetero-dimerize, JAM1 did not interact with MYC2 but it did bind to some promoter regions targeted by MYC2. Therefore, any cellular condition promoting the accumulation of JAM1 or JAM1-like proteins might result in competitive displacement of MYC2 from target JA-responsive genes (Nakata et al. 2013). JAM1 is able to interact with most of the JAZ proteins through its bHLH domain (Song et al. 2013). Two different studies, using multiple *jam* knock-out mutants, clearly established that bHLH IIId transcription factors are negative regulators of the JA-response (Sasaki-Sekimoto et al. 2013; Song et al. 2013). Triple and quadruple *jam* mutants showed a constitutively enhanced JA-related response: *e.g.*, increased accumulation of anthocyanin pigments, JA-hypersensitive roots, delayed flowering, constitutive activation of JA-responsive genes, increased resistance to a necrotrophic pathogen, and increased susceptibility to a biotrophic pathogen (Sasaki-Sekimoto et al. 2013; Song et al. 2013). A transcriptomic study showed that both *JAM1* and *MYC2* are both significantly down-regulated by supplementary FR radiation in JA-exposed plants (de Wit et al. 2013). In addition, FR appeared to enhance the expression of *JAM2* (de Wit et al. 2013). Fluctuations in the MYC: JAM ratio might modulate the strength of the plant defense response as a result of competition between JAM and MYC transcription factors for the promoter regions of common target genes. More work is needed to understand the contribution of JAM1 and other bHLH IIId transcription factors to the repression of JA-mediated immune responses in FR-rich environments.

JAV1: A Novel Family of Transcription Factors

The *JA-ASSOCIATED VQ* motif 1 (*JAV1*) gene was recently found using a library of Arabidopsis RNAi lines showing increased resistance to the necrotrophic fungus *B. cinerea* (Hu et al. 2013). Further characterization showed that *JAV1*-RNAi plants are also more resistant to phytophagous insects. However, in contrast to other JA-signaling elements, JAV1 does not appear to be involved in JA-controlled developmental phenotypes, such as root growth inhibition, anther fertility, and seed production. JAV1 appears to be a negative regulator of the JA pathway (Fig. 1b). JAV1 and JAZ share some common attributes. Thus, as in the case of *JAZ*, *JAV1* gene expression and JAV1 protein turnover are activated by high concentrations of JA-Ile in the cell (for example, as a result of wounding or JA-treatment), and JAV1 degradation requires a functional COI1 protein. Nevertheless, JAV1 does not directly interact with COI1 or JAZ proteins, ruling-out the possibility that JAV1 effects can be mediated through known elements of the JA core signaling module (Hu et al. 2013). Given that JAV1-expression under JA treatment is slightly up-regulated by FR light (de Wit et al. 2013), it is tempting to speculate that

JAV1 contributes to suppress JA responses in FR-enriched environments.

MEDIATOR25/PFT1 Links Transcription Complex to Light Regulation of Defenses

In eukaryotes, the MEDIATOR complex physically connects the basal transcription machinery to transcription regulatory proteins (Conaway and Conaway 2011). Isolation of the plant MEDIATOR complex identified PFT1 (PHYTOCHROME AND FLOWERING TIME 1) as one of its multiple subunits (Bäckström et al. 2007). PFT1 had been discovered initially in a mutant screen for enhanced response to pulses of red light (Cerdán and Chory 2003). Functional studies using *pft1* mutant plants established that PFT1 acts downstream of phytochromes controlling SAS and flowering time responses to low R:FR ratios (Iñigo et al. 2012a, 2012b). PFT1 regulates flowering by controlling the expression of the plant florigen locus, *FLOWERING LOCUS T* and *CONSTANS* (Cerdán and Chory 2003; Iñigo et al. 2012a, 2012b). PFT1 appears to be a molecular hub for light-quality dependent responses and JA-controlled genes (Iñigo et al. 2012a). Interestingly, *pft1* mutant plants showed increased susceptibility to the necrotrophic fungal pathogens *A. brassicicola* and *B. cinerea*, which is consistent with data on PFT1-induced transcriptomic rearrangements (Kidd et al. 2009). It has been recently shown that MED25/PFT1 directly interacts with core transcription factors of the JA-response, like MYC2 and OCTADECANOID-RESPONSIVE ARABIDOPSIS AP2/ERF59 and ERF1 (Fig. 1b) (Cevik et al. 2012), and directly binds to the promoter regions of *JAZ6* and *JAZ8* after JA treatment (Chen et al. 2012). Given the importance of PFT1 in the phyB and defense pathways, it may be relevant to further explore the role of PFT1 in the regulation of JA responses by changes in the R:FR ratio.

NPR1 Phosphorylation and SA Signaling

The protein NPR1 plays a critical role in SA signaling (Fu and Dong 2013). *npr1* mutants are more susceptible to bacterial infection and show no systemic accumulation of PR proteins in response to SA treatment. NPR1 nuclear activity as a transcriptional co-activator is subject to multiple levels of regulation: 1) cytosolic-nuclear partition depending on the cytosolic redox state; 2) expression and accumulation of TGA transcription factors in the nucleus; 3) regulation of NPR1 nuclear protein levels by interactions with NPR3 and NPR4 in the presence of SA; 4) NPR1-phosphorylation, which is necessary for degradation; and 5) NPR1-degradation, which is required for full-induction of NPR1-dependent genes (Fu et al. 2012; Fu and Dong 2013; Mou et al. 2003; Spoel et al. 2009) . A low R:FR ratio has been shown to increase bacterial proliferation when Arabidopsis plants are inoculated with the hemi-biotrophic pathogen *P. syringae* pv. tomato DC3000 (de Wit et al. 2013). A similar result was observed when phyB was inactivated by mutation (Faigón-Soverna et al. 2006; Genoud et al. 2002). Interestingly, recent data have shown that NPR1 levels in the nucleus are higher in plants treated with supplemental FR than in control plants exposed to white light only (Fig. 1a). However, under supplemental FR, NPR1 phosphorylation is attenuated (de Wit et al. 2013). Reduced NPR1 phosphorylation could result from repression of SA-inducible kinases, and be part of a mechanism by which phyB regulates SA signaling and plant resistance to biotrophic pathogens (de Wit et al. 2013).

Concluding Remarks and Perspectives

Plant responses to competitors and attackers are crucial to generate adaptive phenotypes under conditions of variable biotic stress. Negative effects of shading and competition with other plants on plant defense are well documented in the ecological and agricultural literature, but the molecular mechanisms underlying these effects have been elusive. Common interpretations have included a number of resource-based hypotheses, which postulated that the down-regulation of defense under competition is an unavoidable consequence of resource limitation and re-direction of limited resources to other physiological functions (discussed in Ballaré 2014). Although resource limitations would certainly put an upper limit to the investment in defense, it is clear that plants make "decisions" about their defense strategy well before they are starved for resources (or at least for light). As discussed in this review, the active form of phyB (Pfr) is required for full expression of SA- and JA-inducible defenses. Therefore, under conditions of current or potential competition, depletion of the Pfr pool by low R:FR ratios, down-regulate plant immunity against herbivores and pathogens. The connections between phyB and the hormonal pathways that orchestrate plant defense may involve a variety of mechanisms. Some of the molecular pieces of these mechanisms are likely to be regulated by the strength of the R:FR signal. Therefore, in the field, the investment in defense is predicted to be finely modulated by the intensity of the competition signals perceived by plant photoreceptors. Furthermore, because repression of defense responses by phyB inactivation can be localized (*i.e.*, restricted to the plant parts that effectively experience low R:FR ratios) (Izaguirre et al. 2013), photoreceptor-mediated modulation of plant immunity provides a powerful mechanism to optimize the intensity and spatial distribution of the defense effort in patchy and highly dynamic canopy environments. The use of Arabidopsis tools will be critical to map the interactions among the molecular players that generate functional solutions to the growth *vs*. defense allocation dilemma, and can provide important elements and novel genetic markers to be used in crop improvement programs. An

important additional challenge for future research is to determine how the mechanisms of crosstalk between light and hormone signaling that are characterized in the highly tractable Arabidopsis system are modified and regulated under realistic field conditions.

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