Light Regulation of Plant Defense

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Annu. Rev. Plant Biol. 2014. 65:335–63

First published online as a Review in Advance on January 23, 2014

The *Annual Review of Plant Biology* is online at plant.annualreviews.org

This article's doi: 10.1146/annurev-arplant-050213-040145

Keywords

plant immunity, jasmonate, phytochrome, salicylic acid, ultraviolet-B radiation, UVB radiation, UVR8

Abstract

Precise allocation of limited resources between growth and defense is critical for plant survival. In shade-intolerant species, perception of competition signals by informational photoreceptors activates shade-avoidance responses and reduces the expression of defenses against pathogens and insects. The main mechanism underlying defense suppression is the simultaneous downregulation of jasmonate and salicylic acid signaling by low ratios of red:far-red radiation. Inactivation of phytochrome B by low red:far-red ratios appears to suppress jasmonate responses by altering the balance between DELLA and JASMONATE ZIM DOMAIN (JAZ) proteins in favor of the latter. Solar UVB radiation is a positive modulator of plant defense, signaling through jasmonate-dependent and jasmonate-independent pathways. Light, perceived by phytochrome B and presumably other photoreceptors, helps plants concentrate their defensive arsenals in photosynthetically valuable leaves. The discovery of connections between photoreceptors and defense signaling is revealing novel mechanisms that control key resource allocation decisions in plant canopies.

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INTRODUCTION

Biotic stressors play a central role as determinants of plant fitness. Plants must grow fast enough to avoid being outcompeted by their neighbors and, at the same time, need to defend their tissues from the appetites of a huge variety of heterotrophic organisms, including herbivores and microbial pathogens. Adaptive responses to these stressors are often constrained by resource availability. Thus, diversion of resources to growth can limit investment in defense, and allocation to defense can reduce growth and competitive ability against neighboring plants (5, 7, 35, 68). Therefore, plants must strike a precise balance in their responses to these sources of biotic stress.

The mechanisms that plants use to balance resource allocation to deal with biotic stressors are beginning to be understood at the molecular level. Central to the process of efficiently allocating limited resources is the plant's ability to acquire reliable information about the risks posed by competitors and consumer organisms. Not surprisingly, plants have evolved an exquisite diversity of sensory mechanisms to detect biological threats. Light signals, perceived by dedicated photoreceptors, are of paramount importance for the detection of neighboring plants. They provide plants with information about the proximity and activity of other plants and modulate the expression of adaptive morphological and physiological responses (7, 29, 87). Similarly, defense against herbivores and pathogens is based on a highly effective and sophisticated immune system, which is activated upon recognition by specialized receptors that an attack is taking place (75, 85, 130, 160).

How do the signaling mechanisms that orchestrate plant responses to competition and consumer attacks interact to produce an optimal phenotype under different ecological scenarios? Recent work has uncovered strong effects of light signals perceived by photoreceptors on the

expression of plant defenses, providing new insights into the mechanisms that plants use to make adaptive decisions when challenged by multiple biotic stressors.

The purpose of this review is to highlight recent advances in the field of regulation of plant immunity to herbivores and pathogens by informational photoreceptors. I discuss the functional consequences of this regulation in the context of resource allocation trade-offs and their implications for the understanding of plant interactions with other organisms in natural and managed ecosystems. Other recent reviews have covered aspects of the regulation of defense-related processes by light and provide additional perspective on this fast-moving field of research (10, 58, 77, 88, 91, 139).

LIGHT AS A MODULATOR OF PLANT DEFENSE: EVIDENCE FROM FIELD STUDIES

Effects of Shade and Density

Numerous field studies, carried out in the context of ecological questions, have reported descriptions of the effects of leaf shading and increased plant population density (which is usually accompanied by reduced light exposure) on plant interactions with natural enemies. Comparisons of herbivory levels between plants growing in gaps and plants growing beneath the forest canopy are common in the ecological literature (139). Less studied is the influence of shading on disease caused by microbial pathogens, but agronomists and phytopathologists have extensively documented the effects of crop plant density on crop losses to pathogenic microorganisms (26).

Herbivory by canopy arthropods is suppressed in plants grown in full sunlight compared with those grown in shade (139). Based on their analysis of the literature, Roberts & Paul (139) concluded that a common pattern across terrestrial systems is that leaf tissue from plants grown in the shade is more favorable to herbivore growth and/or development, particularly (but not exclusively) for leaf-chewing insects. Similarly, high plant population density often increases insect herbivory in crops (120), and in certain forest ecosystems, thinning is recognized as an effective restoration treatment that reduces the severity of insect attacks (158).

Regarding plant–pathogen interactions, evidence from several studies indicates that shading increases infection by a range of pathogens (139). In addition, high population density has been shown to markedly increase the severity of plant disease in natural ecosystems (4, 15) and agricultural settings (26, 86).

The mechanisms by which shading and crowding increase herbivory and disease severity can be complex and involve multiple causes. For example, in the case of microbial pathogens, microenvironmental factors such as leaf surface wetness (which can be modified by shading) and inoculum transmission (which increases with plant population density) are likely to play an important role. Similarly, herbivorous insects can present direct responses to changes in light levels, which may have an impact on herbivory under natural conditions (111). However, several studies have shown that infection by a range of pathogens and the success of several insect herbivores can be affected by the light environment of the host before contact with the consumer organism (10, 139), indicating that light can have important effects on plant defense. The general pattern that has emerged from these studies is that plant resistance to pests and pathogens is often weakened by leaf shading or increased canopy density.

Historical Interpretations

Several theories have been developed to explain variations in resource allocation to defense as a function of environmental factors, including light conditions. In general, these classic models

of plant defense postulate that allocation to carbon-rich secondary metabolites (including plant defenses), such as phenolic compounds and terpenes, increases as a function of the surplus of carbohydrates that results when photosynthetic CO_2 fixation exceeds carbohydrate utilization for growth. Examples include the carbon/nutrient and growth/differentiation balance hypotheses (25, 68). These so-called resource-based hypotheses have been shown to lack generality (65, 89, 99), and, more important, they do not explain, in mechanistic terms, how the shifts in allocation are effectively implemented within the plant. In spite of these limitations, these theories are still frequently used in the design and interpretation of ecological experiments.

Biotroph: a pathogen that feeds on living host cells

Necrotroph: a pathogen that feeds on nutrients released by dead host cells

Major advances in the field of plant biology have produced, in the past 20 years, a core model of defense regulation in plants, which is based on the interactions of several key hormonal players. This model provides a general framework to interpret environmental regulation of defense, based on the connections between environmental variables (such as light) and the major regulatory hubs that control the plant immune responses.

The following two sections provide a basic outline of our current understanding of the plant immune system and a brief description of the mechanisms used by plants to detect changes in their light environment. After these introductory sections, I concentrate on the roles of specific photoreceptors in regulating defense signaling.

PLANT IMMUNITY AGAINST HERBIVORES AND PATHOGENS

Pathogens and Herbivores: Some Basic Concepts and Definitions

The combined action of herbivorous animals (mostly insects) and microbial pathogens accounts for approximately 25% of preharvest crop losses in areas with well-developed agricultural practices (125). Microbial pathogens are usually classified as biotrophs or necrotrophs according to their lifestyles. Biotrophs invade the plant and feed on the contents of living tissues, causing minimal damage to the host cells, whereas necrotrophs first kill the cells, causing significant tissue damage, and then feed on the remains (60). Among animal herbivores, phytophagous arthropods are the major plant consumers in many ecosystems as well as in agricultural systems (144). Arthropod herbivores are also classified according to their feeding strategies, with tissue chewers and pierce-suckers being the principal guilds in most ecosystems.

Plants defend themselves against consumer organisms using a combination of direct and indirect defenses. Direct defenses target the attacking organism and are frequently based on the deployment of molecules that either deter or interfere with the metabolism of the invader. Indirect defenses, which are highly effective against herbivores, are based on the production of substances or structures that attract natural enemies of the plant consumer, such as specific blends of volatile compounds and extrafloral nectar (89, 144). Both direct and indirect defenses can be inducible (i.e., activated in response to attack).

Plant Immune Responses Against Herbivores and Pathogens

In contrast to vertebrates, plants do not have specialized, mobile immune cells circulating through their bodies in search of potential enemies. However, plants are able to produce highly effective and specific immune responses and to generate mobile signals that activate specific defenses in tissues away from the primary site of infection, thus providing systemic resistance to the attacker. The plant immune system (**Figure 1*a***), like the innate immune system of animals, is activated upon recognition of non-self (or damaged-self) molecular patterns or signals (75, 85, 130, 160).

Recognition of attack. Microbial plant pathogens are recognized by two types of receptors: pattern-recognition receptors (PRRs) and intracellular resistance (R) proteins (**Figure 1*a***). PRRs are located on the cell surface and are activated by conserved pathogen-associated molecular patterns (PAMPs), such as flagellin and chitin, or by damage-associated molecular patterns (DAMPs), which are molecules that result from damage to plant tissue. PRR activation triggers a first layer of plant defense known as PAMP-triggered immunity. Pathogens can often overcome this first layer of defense, for example, by using effector molecules that inactivate PAMP-triggered immunity. Resistant plants activate a second line of defense, known as effector-triggered immunity, which is triggered when polymorphic intracellular R proteins recognize attacker-specific effectors and then renders the pathogen avirulent. Nearly all intracellular immune receptors belong to the nucleotide-binding site–leucine-rich repeat protein family (55, 85, 127, 130).

Insect herbivores are detected by the perception of DAMPs as well as herbivore-associated molecular patterns (HAMPs) (19, 47, 67, 75, 160) (**Figure 1*a***). Multiple HAMPs have already been identified, including fatty acid–amino acid conjugates (FACs) (2), caeliferins, inceptin, oligouronides, glucose oxidase, β-glucosidase, and lipases (19, 47). FACs are probably the best-characterized insect elicitors and are present in the oral secretions of most lepidopteran larvae (170). They are synthesized from plant-produced fatty acids (such as linolenic and linoleic acids), which are conjugated with glutamine or glutamic acid. Because FACs do not occur in undamaged plants, they provide a reliable signal of herbivore attack. The plant receptors for FACs (as well as those for all other HAMPs) remain to be elucidated, and whether these receptors directly recognize the putative HAMP molecules or some of their metabolic derivatives is unclear (19). Herbivorous insects can also produce molecules to suppress induced defenses, a strategy that has functional analogies to that activated by microbial effectors (19, 47, 71).

Jasmonate (JA): a hormone essential for plant immune responses against necrotrophic pathogens and herbivorous insects

Salicylic acid (SA): a hormone essential for plant immune responses against biotrophic pathogens

Defense hormones: the jasmonate–salicylic acid backbone. After the initial detection of molecular signals of a potential consumer, various hormones coordinate the activation of an immune response (**Figure 1*a***). Jasmonate (JA)—which comprises jasmonic acid and related molecules—and salicylic acid (SA) are the central signaling players in the orchestration of plant defense and form the hormonal backbone of the plant immune system. Major breakthroughs have been made in recent years in elucidating the mechanisms of perception of these hormones, including the characterization of the receptors for JA (31, 155) and SA (56, 162). Below, I briefly outline some key features of the JA-SA backbone that are important for understanding the mechanisms of regulation of plant immunity by light signals. Several recent reviews have described these features in considerably more detail (23, 52, 55, 126, 129, 130).

Salicylic acid. The SA response pathway is activated predominantly to fend off biotrophic microbial pathogens (60). SA biosynthesis is triggered in the chloroplast during PAMP-triggered immunity and effector-triggered immunity upon recognition of microbial signatures (PAMPs or microbial effectors). Although the principal biosynthetic enzymes have been characterized, as have some of the early signaling events, how pathogen recognition induces the synthesis of SA, both locally and systemically, is not entirely clear (55, 130).

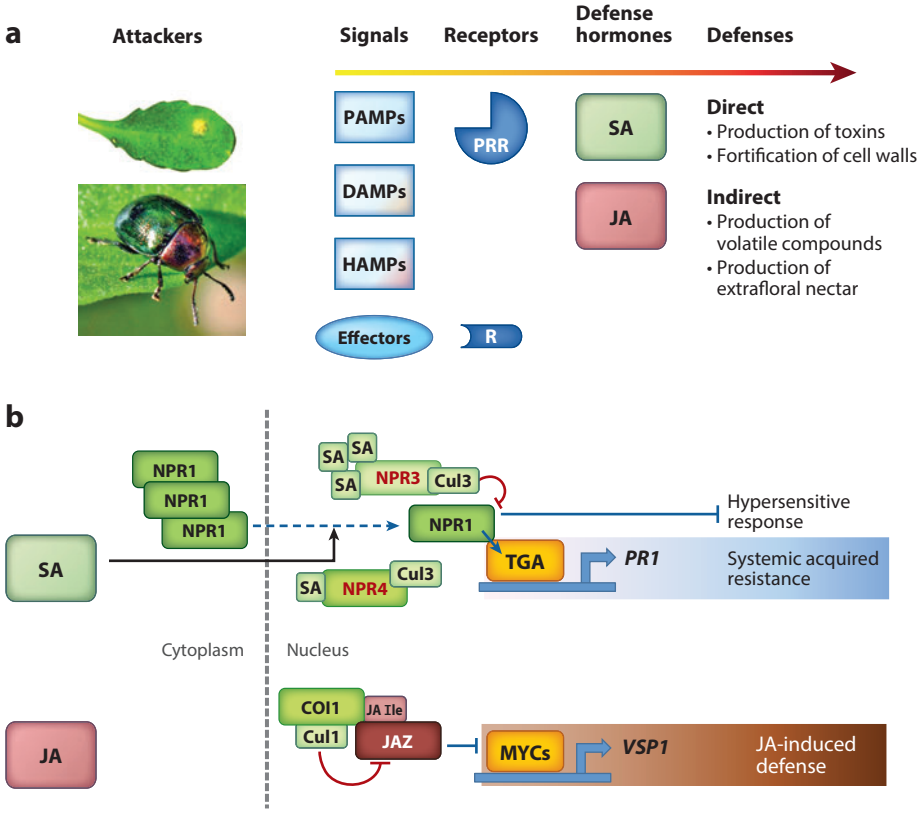
Effector-triggered immunity is often associated with programmed cell death at the infection site (hypersensitive response), which is a simple and effective mechanism by which plants can isolate and inactivate biotrophic pathogens (55). An avirulent pathogen not only triggers defense responses at the site of invasion but also activates SA biosynthesis and the production of mobile molecules such as methyl-SA, azelaic acid, glycerol-3-phosphate, and abietane diterpenoid dehydroabietinal, which leads to systemic expression of a large set of defense-related genes in other tissues (55). These

Systemic acquired resistance: a broad-spectrum plant disease resistance

NONEXPRESSOR OF *PR* GENES 1 (NPR1): a protein that is required for *PR* gene expression, local defense, SA signaling, and systemic acquired resistance

genes include *PATHOGENESIS RELATED* (*PR*) genes (some of which encode proteins with antimicrobial activities) and genes encoding several WRKY transcription factors (130). This systemic response—which protects the rest of the plant from secondary pathogen invasion, requires SA, and confers broad-spectrum resistance—is referred to as systemic acquired resistance (55, 130, 157).

The SA receptor has been elusive, but recent work is beginning to shed light on its molecular identity (**Figure 1*b***). The protein NONEXPRESSOR OF *PR* GENES 1 (NPR1) has long been recognized as a central regulator of SA-induced responses (55). NPR1 is required for systemic acquired resistance but can repress the hypersensitive response caused by effector-triggered immunity (55, 135). In the presence of SA, inactive NPR1 oligomers in the cytosol are monomerized owing to SA-induced changes in the redox state of the cell and then translocated to the nucleus. Once in the nucleus, NPR1 monomers function as coactivators of TGA transcription factors that regulate the expression of SA-responsive, defense-related genes, including *PR* genes (42, 55, 126). Fu et al. (56) recently proposed that two NPR1 paralogs, NPR3 and NPR4, are SA receptors that regulate NPR1 levels as a function of SA concentration in the tissue and, consequently, cell fate and defense (i.e., no defense activation, hypersensitive response, or systemic acquired resistance) (**Figure 1*b***). However, evidence from another recent study (162) indicates that NPR1 can directly bind SA and supports a role for NPR1 as the bona fide SA receptor. NPR1 and NPR3/NPR4 may work as a multireceptor complex, and additional work is needed to establish the precise roles of these proteins in SA perception (126).



Jasmonate. In response to attack by necrotrophic pathogens or chewing insects, which often cause tissue disruption and death of multiple cells, plants activate the JA response pathway. This pathway begins with the release of α-linolenic acid from chloroplast membrane galactolipids, leading to the production of jasmonic acid through a well-characterized biosynthesis pathway (159). Jasmonic acid can then be conjugated to amino acids such as isoleucine to form the bioactive hormone, an enantiomer of jasmonoyl-isoleucine (JA-Ile) (53). Induction of the JA pathway leads to massive changes in gene expression and activation of direct and indirect defenses (75). After the JA pathway is activated at the initial site of damage or herbivory, long-distance signaling mechanisms induce JA responses in other tissues and organs, providing systemic protection against future attacks (75).

JASMONATE ZIM DOMAIN (JAZ) proteins: proteins that play a central role as repressors of the JA signaling pathway by targeting several key JA-responsive transcription factors

Perception of JA-Ile is achieved by a coreceptor formed by the ubiquitin E3 ligase complex Skp1-Cul1-F-box protein CORONATINE INSENSITIVE 1 (SCF^{COI1}) and JASMONATE ZIM DOMAIN (JAZ) proteins (23, 52) (**Figure 1*b***). JA-Ile stimulates the specific binding of COI1 and JAZ proteins, which leads to ubiquitination of JAZs by SCF^{COI1} and their subsequent proteasome-mediated degradation (31, 116, 128, 146, 155, 166, 167). JAZs are repressors of transcription factors that are positive regulators of JA responses. Therefore, degradation of JAZs triggers the activation of JA-induced defense (92, 129). Among the targets of JAZs that activate JA-controlled defenses in *Arabidopsis* are transcription factors that regulate (*a*) resistance to insects, such as the basic helix-loop-helix (bHLH) proteins MYC2, MYC3, and MYC4 (51); (*b*) anthocyanin biosynthesis and trichome initiation, such as the bHLH (TT8, GL3, and EGL3) and MYB (MYB75 and GL1) proteins that are essential components of WD-repeat/bHLH/MYB

Figure 1

Overview of the plant immune system. (*a*) Schematic representation of the series of events leading to defense activation. When plants are attacked by pathogens or herbivores, different types of receptors, including pattern-recognition receptors (PRRs) and resistance (R) proteins, recognize non-self (or damaged-self) molecular patterns or signals, including pathogen-, damage-, and herbivore-associated molecular patterns (PAMPs, DAMPs, and HAMPS, respectively), as well as effector molecules. Downstream of the initial perception, two key defense hormones, salicylic acid (SA) and jasmonate (JA), coordinate the expression of immune responses, leading to defense activation. Plant defenses can be direct (those that directly target the consumer organism, such as production of toxins and fortification of cell walls) or indirect (those that attract natural enemies of the attackers). Among the latter, production of volatile compounds and production of extrafloral nectar are the best characterized. (*b*) Models of SA and JA perception. (*Top*) In the presence of SA, inactive NONEXPRESSOR OF *PR* GENES 1 (NPR1) oligomers in the cytosol are monomerized and translocated to the nucleus. Once in the nucleus, NPR1 activates TGA transcription factors that regulate the expression of SA-responsive genes, such as *PR1* genes. According to the model described by Fu & Dong (55; see also 126 and 162), the nuclear NPR1 concentration is controlled by SA through the SA receptor proteins NPR3 and NPR4, which directly interact with the E3 ligase Cul3 and function as adaptors to mediate NPR1 degradation. SA facilitates the interaction between NPR1 and $Cul3^{NPR3}$ but disrupts the interaction between NPR1 and $Cul3^{NPR4}$. At the site of infection, under high SA levels, NPR1 is degraded by $Cul3^{NPR3}$, allowing programmed cell death (hypersensitive response). In neighboring cells, SA levels are intermediate—not high enough to promote interaction between NPR1 and $Cul3^{NPR3}$ but sufficient to disrupt the interaction between NPR1 and $Cul3^{NPR4}$. Under these conditions, NPR1 concentration increases, inhibiting the hypersensitive response and triggering systemic acquired resistance. (*Bottom*) Perception of jasmonoyl-isoleucine (JA-Ile) is achieved by a coreceptor formed by the Skp1-Cul1-F-box protein COI1 (SCF^{COI1}) complex (for simplicity, only Cul1 and COI1 are shown) and JAZ. JA-Ile stimulates the binding of COI1 and JAZ proteins, which leads to ubiquitination of JAZ proteins by SCF^{COI1} and their subsequent proteasome-mediated degradation. JAZ proteins are repressors of transcription factors that are positive regulators of JA responses, such as MYC2. Therefore, degradation of JAZ proteins triggers the activation of JA-induced defense [shown here as an example is *VEGETATIVE STORAGE PROTEIN1* (*VSP1*)]. Solid arrows indicate positive interactions; truncated connectors indicate negative regulation. The dashed arrow denotes the translocation of NPR1 after SA-promoted monomerization, which is indicated by the black arrow.

complexes (132); and (*c*) JA–ethylene interactions and resistance to necrotrophic pathogens, such as ETHYLENE INSENSITIVE 3 (EIN3) and EIN3-LIKE 1 (EIL1) (172). Recent research has characterized various bHLH transcription factors that are also targets of JAZ proteins but act as transcriptional repressors (123, 143, 148). Although not yet explicitly tested in physiological experiments, coordinated regulation of JA responses by transcription activators and repressors is likely to allow fine regulation of defense responses. Other points of regulation of JA responses may include modulation of COI1 levels by SCF^{COI1} (165) and JA-ASSOCIATED VQ MOTIF (JAV1), a recently characterized protein that is degraded in a COI1-dependent manner via the 26S proteasome pathway in response to JA and functions as a repressor of JA responses and insect and pathogen resistance (76).

The jasmonate–salicylic acid backbone and hormonal crosstalk. The JA-SA backbone (**Figure 1**) provides a conceptual framework to study the regulatory roles of internal and external signals in plant defense. That JA signaling and SA signaling can antagonize each other is well established (21, 102, 130); plants infected by biotrophic pathogens often suppress JA-dependent signaling (49, 149), and, conversely, activation of the JA pathway can repress SA responses (22, 156). This mutual antagonism between the JA and SA pathways has been interpreted as a mechanism by which plants can intelligently focus their defense strategies on the right consumer targets (8, 130).

The influence of other internal signals, notably other plant hormones, in the regulation of these major defense pathways has attracted considerable attention. In particular, the role of the gaseous hormone ethylene is well characterized (130). Hormonal crosstalk has emerged as a major research focus in recent years, and considerable evidence indicates that plant hormones not traditionally linked with defense responses, such as auxins, gibberellins (GAs), and cytokinins (CKs), can play major regulatory roles in JA- and SA-mediated defense activation (8, 47, 130, 138). Because the levels of all of these hormones can be modulated by light signals perceived by photoreceptors, this modulation must be taken into account when considering the effects of canopy light on JA and SA responses.

GA: gibberellin

CK: cytokinin

UV RESISTANCE LOCUS 8 (UVR8): a protein that functions as a specific UVB photoreceptor in *Arabidopsis*

Phytochrome B (phyB): a member of the phytochrome photoreceptor family that regulates SAS responses to low R:FR ratios

Red:far-red (R:FR) ratio: the ratio between the photon irradiances (μmol m^{-2} s^{-1}) at 660 and 730 nm

PERCEPTION OF COMPETITORS: LIGHT SIGNALS AND PHOTORECEPTORS

The principal photoreceptors involved in the perception of competitors are the phytochromes, cryptochromes, and possibly phototropins and UV RESISTANCE LOCUS 8 (UVR8) (**Figure 2**).

Phytochrome B

The main photoreceptor used by plants to detect the proximity of competitors is phytochrome B (phyB), as demonstrated by experiments employing *phyB* mutants in real plant canopies (6). Chlorophyll-containing plant tissues absorb strongly in the visible region of the solar spectrum, which includes blue (B) and red (R) light, whereas far-red (FR) photons are either transmitted or reflected. Therefore, light transmitted through or reflected by a plant canopy has a low R:FR ratio compared with sunlight, which has an R:FR ratio of approximately 1.2 (147). Under high R:FR, the phyB photoreceptor resides in the nucleus, predominantly in its active form (Pfr), preventing the accumulation and activity of growth-promoting bHLH transcription factors known as PHYTOCHROME-INTERACTING FACTORS (PIFs) (83, 104). When the R:FR ratio is lowered by the proximity of vegetation, phyB is partially inactivated, as the levels of phyB Pfr are reduced by FR-promoted photoconversion into the inactive form of the photoreceptor (Pr) (147). In *Arabidopsis* seedlings, reduction of phyB Pfr levels results in increased activity of

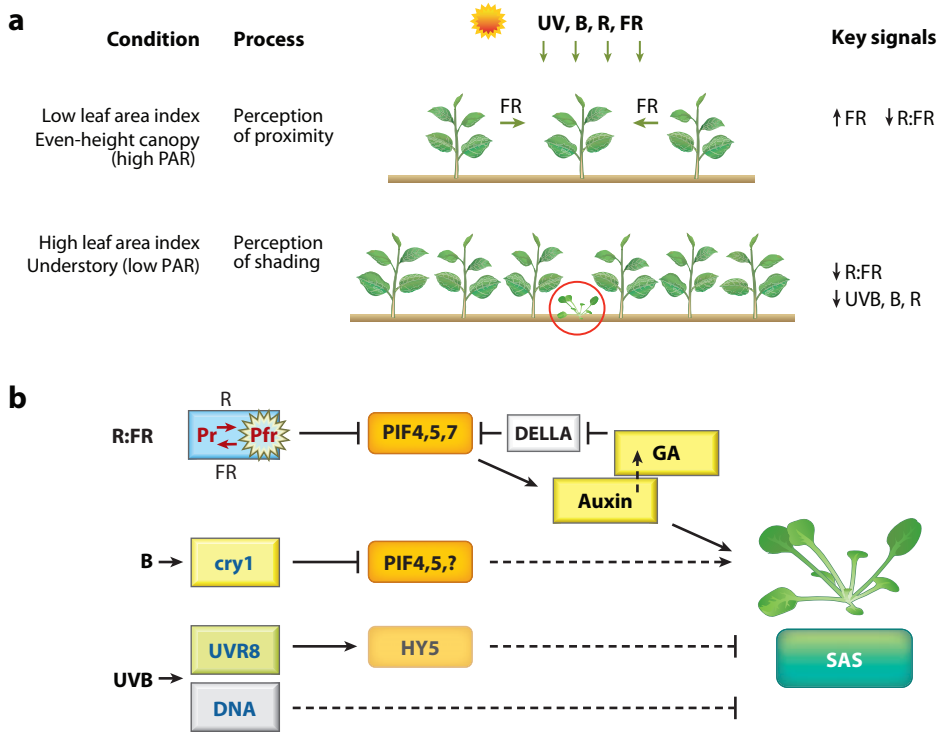


Figure 2

Perception of and response to competition with other plants. (*a*) Schematic representation of two scenarios of neighbor perception. (*Top*) Perception of proximity. In even-height stands, plants can detect the threat of competition posed by surrounding vegetation by perceiving far-red (FR) radiation reflected by neighboring plants. Under these conditions, the key light signal of plant proximity is the increase in FR radiation [reduction of red (R):FR ratio], which is perceived by the photoreceptor phytochrome B (phyB). (*Bottom*) Perception of shading. At a high leaf area index (LAI), or when emerging underneath an established canopy (denoted by the seedling inside the red circle), plants perceive actual shading using a variety of light signals, which include reduced R:FR ratio and reduced irradiances of UV, blue (B), and R light. Several photoreceptors participate in the perception of light quality and quantity under these conditions, including phytochromes, cryptochromes, and UVR8. Phototropins participate in the perception of directional light signals and foraging for light in canopies, but because they do not have well-defined roles in plant defense, they are not included in this figure. Additional abbreviation: PAR, photosynthetically active radiation. (*b*) Mechanisms behind activation of the shade-avoidance syndrome (SAS). SAS responses triggered by low R:FR are the best understood. Low R:FR ratios reduce the levels of the active (Pfr) form of the photoreceptor phyB, which leads to increased activity of several PHYTOCHROME-INTERACTING FACTORS (PIFs). PIFs are growth-promoting transcription factors that activate genes involved in auxin biosynthesis. Increased levels of auxin lead to faster stem and petiole elongation. Low R:FR ratios can also produce increased levels of gibberellins (GAs). GAs trigger the degradation of DELLA proteins, which are repressors of PIFs. Under conditions of actual shade, when B-light levels are attenuated, reduced activation of the photoreceptor cryptochrome 1 (cry1) also leads to strong SAS responses. The mechanisms are not well understood, but genetic evidence also indicates a role for PIFs in SAS responses to B-light depletion. Reductions in UVB levels also trigger SAS-like responses through poorly characterized mechanisms. The UVR8 photoreceptor is involved in morphological responses to UVB attenuation. Because the transcription factor HY5 participates in UVR8-induced photomorphogenesis, it is likely involved in UVR8-mediated responses to shading. Attenuation of UVB radiation can reduce the levels of DNA damage, which could result in increased growth in shaded canopy positions. Arrows indicate positive interactions; truncated connectors indicate negative regulation. Dashed lines denote regulation for which there is no direct empirical evidence or for which the biochemical mechanism is unclear. Additional abbreviation: Pr, inactive form of phyB.

PIF4, PIF5 (107), and PIF7 (105), which promotes the expression of genes encoding enzymes involved in the synthesis of auxin (72, 105, 154). Increased levels of auxin are transported to the hypocotyls (72, 96), leading to faster cell elongation (**Figure 2*b***). In more mature plants of several species, the suite of morphological changes triggered by phyB inactivation often includes increased elongation of stems and petioles, apical dominance, and production of erect leaves. Collectively, this morphological reconfiguration is known as the shade-avoidance syndrome (SAS) (7, 29, 87), and it is an essential determinant of plant competitive ability in crowded populations (7). PIFs also promote the expression of negative regulators of SAS, such as several HLH proteins, which are thought to play a role in fine-tuning elongation responses to shade-light signals (57, 73). The hormonal regulation of SAS in plants that have grown beyond the seedling stage is likely to be more complicated than the one described for the *Arabidopsis* seedling model. For example, hypocotyl elongation appears to be controlled by hormonal signals derived from the cotyledons, whereas in more mature plants, the stems themselves can perceive changes in R:FR ratio and adjust elongation growth according to the local light environment (11, 121). In fact, an important feature of the R:FR ratio as a proximity signal is that it allows the stems and petioles to detect lateral radiation reflected by potential competitors and activate SAS responses before the plants are severely shaded by neighboring vegetation (12) (top section of **Figure 2*a***).

Shade-avoidance syndrome (SAS): a suite of morphological changes expressed by plants when grown at high population density or under the shade of other plants; it typically includes increased stem elongation and strong apical dominance

Leaf area index (LAI): the ratio between one-sided green leaf area and ground area; values vary from 0 (for bare soil) to >10 (for dense, closed canopies)

UVB radiation: the region of the electromagnetic spectrum between 280 and 315 nm

Cryptochromes and UVR8

When neighboring plants are close enough to each other to cause a significant degree of mutual shading—e.g., at a high canopy leaf area index (LAI)—or when a plant becomes shaded by taller competitors, other light cues and photoreceptors become important in competition signaling (bottom section of **Figure 2*a***). Under those conditions, even *phyB* null mutants show robust responses to crowding and shading (95). Cryptochromes can elicit typical SAS responses when the plants become exposed to low levels of B light (95, 97, 145). The mechanisms that mediate these effects are not completely clear, but genetic evidence indicates that *PIF4* and *PIF5* play a functional role in the activation of SAS responses elicited by B-light depletion (95) (**Figure 2*b***).

Under high-density conditions and significant shading, the dose of UVB radiation received by individual plants is also significantly reduced (61). Attenuation of solar UVB radiation can have subtle effects promoting elongation growth, which may be caused by alleviation of toxic effects of solar UVB (such as reduction in the load of DNA damage) (109) or photomorphogenic effects resulting from reduced activation of the specific UVB photoreceptor UVR8 (40, 66) (**Figure 2*b***). Because the characterization of UVR8 as a UVB photoreceptor is a relatively recent advance (137) and the downstream signaling cascades are still not fully characterized (66, 82), we are far from having a complete picture of the role of this photoreceptor in mediating SAS responses in real canopies.

PHOTORECEPTORS AND PLANT DEFENSE

A growing body of evidence indicates that photoreceptor-mediated perception of shading or neighbor-proximity signals modulates the expression of plant immune responses (**Figure 3**). Changes in the R:FR ratio (perceived by phyB) and UVB radiation (presumably acting through UVR8) are the canopy light signals whose influences on plant resistance to herbivores and pathogens have been best documented. The following sections review the evidence for adaptive modulation of plant immunity by R:FR and UVB radiation.

Phytochrome and Red:Far-Red Ratio

Plants exposed to light with a low R:FR ratio express a weak defense phenotype when tested in bioassays. This was initially demonstrated in experiments using *Nicotiana longiflora* plants growing

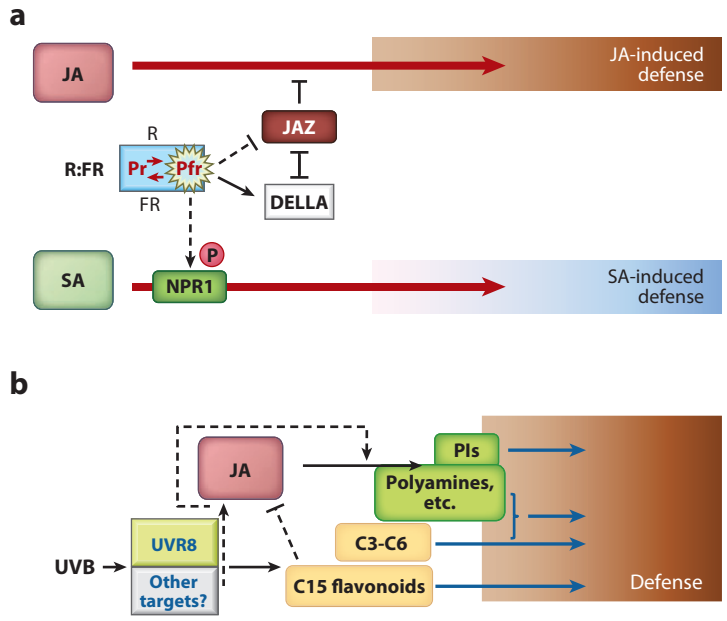


Figure 3

Photoreceptors and plant defense. (*a*) Phytochrome B (phyB) is a positive regulator of jasmonate (JA) and salicylic acid (SA) signaling. Low red:far-red (R:FR) ratios, which reduce the levels of the active form of phyB (Pfr), downregulate JA responses, presumably via mechanisms that involve antagonistic interactions between DELLA and JAZ proteins, and repress SA-induced defenses through unknown mechanisms that might involve reduced NPR1 phosphorylation. (*b*) UVB radiation enhances defense responses through JA-dependent and JA-independent signaling mechanisms. Acting through UVR8, solar UVB radiation promotes the synthesis of phenolic compounds that can act as direct defenses against herbivores and pathogens. In addition, UVB can increase JA synthesis and sensitivity. Neither the photoreceptors nor the signaling intermediates for the effects of UVB radiation on JA responses have been identified. In solanaceous species, increased JA signaling in response to solar UVB radiation can lead to higher expression of proteinase inhibitors (PIs), which are important direct defenses, and the biosynthesis of certain secondary metabolites (such as polyamines) that, conjugated with phenylpropanoids (C3-C6), can have antiherbivore functions. Phenylpropanoids and flavonoids can have direct roles in defense, and, in *Arabidopsis*, flavonoids can repress accumulation of bioactive JA. Arrows indicate positive interactions; truncated connectors indicate negative regulation. Dashed lines denote regulation for which the molecular mechanisms are unclear. Additional abbreviation: Pr, inactive form of phyB.

under sunlight or sunlight supplemented with FR radiation to mimic the proximity of neighboring plants without altering photosynthetically active radiation (PAR) or photosynthesis (simulating the scenario depicted in the upper section of **Figure 2*a***). When those plants were used in insect-feeding experiments, caterpillars of the specialist herbivore *Manduca sexta* grew much faster on FR-supplemented plants than on plants that received ambient light only (80). Similar results were subsequently reported for *Arabidopsis* when plants were exposed to equivalent light treatments and tested in bioassays using insect herbivores (*Spodoptera frugiperda*) (120) and microbial pathogens, including biotrophs (*Pseudomonas syringae* pv. *tomato* DC3000) (38) and necrotrophs (*Botrytis cinerea*) (30, 38). Comparable results (increased susceptibility to insects and pathogens) have been obtained in experiments in which phyB was inactivated by mutation (30, 38, 48, 91). Most of the studies on *phyB* mutants were carried out using *Arabidopsis* under controlled light

Photosynthetically active radiation (PAR): photon irradiance between 400 and 700 nm; it has a typical peak value of 2,000 μmol m^{-2} s^{-1} on clear summer days at midlatitudes

conditions; however, experiments with *phyB* mutants of tomato (80) and cucumber (113) provided additional validation of the phenomenon under field conditions.

The effect of phyB inactivation increasing plant susceptibility to pathogens and herbivores is not simply a by-product of prioritizing resource allocation to the generation of the SAS phenotype. *Arabidopsis* mutants that fail to induce SAS but have otherwise normal phytochrome responses, such as the *sav3* mutant (154), still show increased susceptibility to insects and pathogens when exposed to low R:FR ratios (30, 120). Similarly, triggering *Arabidopsis* plants to express a SAS phenotype by manipulating other photoreceptors, such as cryptochrome 1 (cry1), activates increased elongation responses (95) but does not result in a weak defense phenotype in *B. cinerea* infection bioassays (30).

In the following sections, I discuss recent results showing that phyB is a positive regulator of the principal hormonal pathways that orchestrate plant immune responses (**Figure 3*a***).

Phytochrome and jasmonate. Inactivation of phyB by low R:FR ratios or mutation leads to reduced expression of the JA signaling pathway. The effect of low R:FR ratios, repressing JA responses, was originally demonstrated in experiments in which a slight reduction in the R:FR ratio, simulating the proximity of neighboring plants in canopies of low LAI, caused a marked reduction in the sensitivity of *Arabidopsis* plants to exogenous methyl-JA applications (120). Similar effects (repression of JA sensitivity) were observed in the *phyB* mutant (120). The original experiments of Moreno et al. (120) measured only a few JA-responsive marker genes (such as *ERF1* and *PDF1.2*), but the generality of their conclusions has been corroborated by global gene expression profiling experiments (30, 38). Furthermore, de Wit et al. (38) showed that when *Arabidopsis* plants are exposed simultaneously to methyl-JA and supplemental FR radiation, low R:FR ratios repress the expression of JA response markers, but methyl-JA fails to reduce the expression of SAS response markers (such as *PIL1*) and to eliminate petiole elongation responses to low R:FR. Collectively, these results suggest that when shade-intolerant *Arabidopsis* plants are simultaneously exposed to competition and consumer attack signals, responses to low R:FR take priority over responses to JA. Emerging evidence from laboratory and field studies suggests that light environments rich in FR radiation (133) or vegetation shade (1) can also reduce the accumulation of bioactive JA.

The effect of low R:FR reducing JA sensitivity is not limited to effects on transcription: Repression of the accumulation of metabolites covering virtually the whole spectrum of JA-induced plant defenses has been demonstrated (10). In *Arabidopsis* and other members of the mustard family, low R:FR ratios reduce the accumulation of soluble phenolics, anthocyanins, and glucosinolates (30, 46, 120). Furthermore, even JA-induced indirect defenses were drastically reduced by low R:FR ratios in two recent studies. One report demonstrated repression of JA- and wound-induced extrafloral nectar production in passion fruit (*Passiflora edulis*) plants supplemented with FR radiation under high PAR (79), and another study indicated that the emissions of both constitutive and JA-induced green leaf volatiles and terpenoids in *Arabidopsis* were partially suppressed under low R:FR and severe shading conditions (93).

Evidence from field experiments also suggests that conditions of shading or high population density result in attenuated JA responses. For example, milkweed plants (*Asclepias syriaca*) responded to herbivory by monarch caterpillars with increased production of latex—a defense chemical that is JA inducible (134)—but this response was completely eliminated if the plants were exposed to shade from vegetation before the herbivory treatment (1). In another study, ponderosa pines (*Pinus ponderosa*) responded to forest-thinning treatments with increased resin production (158), and wound-induced resin production in conifers is controlled by JA (108).

To summarize, patterns emerging from controlled-environment and field studies involving a broad range of plant taxa support the general hypothesis that the effects of low R:FR ratios reducing plant resistance to necrotrophs and chewing insects are mediated by downregulation of

the JA response pathway, which leads to decreased production of direct and indirect defenses. Downregulation of JA responses by low R:FR ratios has also been observed in recent studies of plant interactions with beneficial bacteria (153).

DELLAs: proteins that repress GA responses by targeting GA-responsive transcription factors

Mechanisms of phytochrome B–jasmonate crosstalk. The molecular mechanisms that explain these effects of low R:FR ratio on JA sensitivity are still unclear. Regulation of plant defense by phyB has been recently linked to JAZ activity, because the effect of low R:FR ratios reducing *Arabidopsis* resistance to *B. cinerea* is significantly attenuated in genotypes disrupted in the expression of the *JAZ10* gene (30). Furthermore, in contrast to the effect of SA as a repressor of the JA response, FR requires a functional SCF^{COI1}-JAZ module to suppress JA-induced gene expression (30).

A possible mechanism for light regulation of JA action may involve phyB-mediated changes in *JAZ* gene expression or JAZ protein stability. Increased expression of certain *JAZ* genes has been observed in response to low R:FR ratios (120). A phytochrome effect on JAZ stability was demonstrated for phytochrome A (phyA), as active phyA appeared to be required for COI1-mediated degradation of JAZ1 (140). However, it remains to be demonstrated whether in fully de-etiolated plants, where responses to low R:FR are controlled predominantly by phyB (6), changes in the levels of phyB Pfr in response to R:FR affect the turnover of JAZ repressors. Recent work has shown that low R:FR ratios increase the stability of JAZ10 and that *jaz10* null mutants fail to display the characteristic effects of low R:FR repressing growth and defense responses to methyl-JA (M. Leone, M.M. Keller & C.L. Ballaré, unpublished observations). These results suggest that the effects of low R:FR inhibiting JA responses in *Arabidopsis* are mediated by a reduction in JAZ10 turnover (**Figure 4**). Stable variants of JAZ proteins produced by alternative splicing can desensitize JA responsiveness (33, 34, 167). Overexpression of JAZ10.4, an alternatively spliced form of JAZ10 that is resistant to COI1-mediated degradation, suppresses JA responses (34). Whether the effect of FR on JA sensitivity is mediated by changing the relative abundance of stable JAZ variants remains unclear. The availability of *Arabidopsis* lines in which the various splice variants of JAZ10 are expressed from the native *JAZ10* promoter in the *jaz10* mutant background (119) provides tools to understand the potential role of these proteins in attenuating JA signaling under low R:FR ratios.

Changes in R:FR could affect JA responses by regulating the levels of other hormones that interact with JA signaling, including SA, GAs, auxin, and brassinosteroids (8, 47, 130, 138). The possibility that increased SA signaling could mediate the effect of low R:FR ratios repressing JA responses has been ruled out (30, 38). GAs often display increased activity in response to neighbor proximity and low R:FR ratios (44, 103). GAs promote growth by triggering proteasome-mediated turnover of DELLA proteins (69), which inhibit elongation by repressing the transcriptional activity of PIFs (37, 50). Interestingly, GAs are known to antagonize certain JA responses (124) and vice versa (169). DELLAs are positive regulators of JA signaling, binding to JAZs and preventing them from repressing JA-responsive transcription factors (74). Therefore, increased GA levels lead to increased turnover of DELLAs and stronger repression of the JA response by JAZs. Interestingly, recent work indicated that JAZs can also affect DELLA function, by blocking the interaction between DELLAs and PIFs (169). The fact that DELLAs and JAZs mutually antagonize their ability to interact with downstream transcription factors could explain the negative interactions between GA and JA signaling, and provides a plausible mechanism by which low R:FR ratios could lead to weaker JA responses (**Figure 4**).

Auxin is another growth-promoting hormone, and it plays a central role in triggering SAS responses in *Arabidopsis* (72, 96, 105, 118, 141, 154). Auxin interacts with JA responses, although conflicting results have been reported regarding the direction of the interaction (i.e., promotion or repression) (138). Auxin can promote GA synthesis (54), which might reduce JA signaling

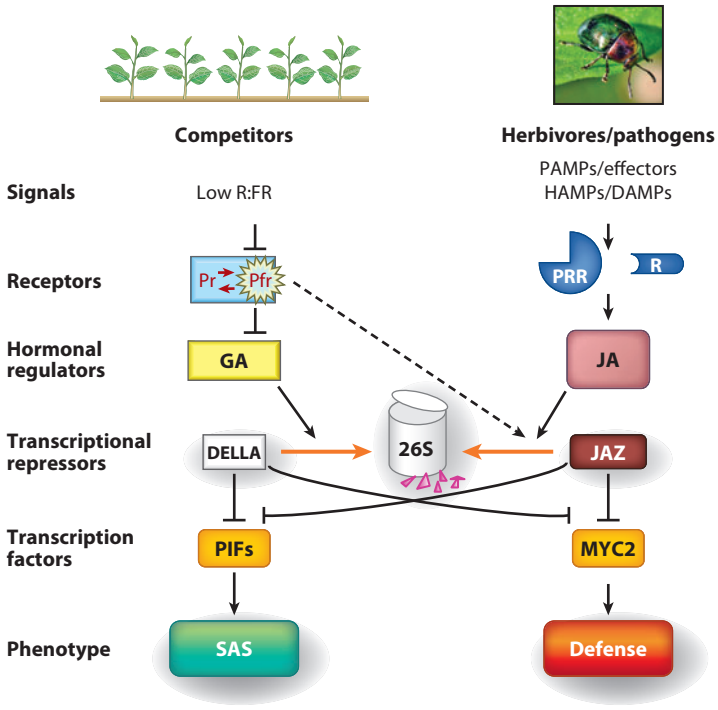


Figure 4

Effects of low red:far-red (R:FR) ratios on jasmonate (JA) sensitivity. Acting via inactivation of phytochrome B (phyB), low R:FR ratios downregulate JA-induced defenses by shifting the balance between DELLA and JAZ repressors. Low R:FR ratios result in increased activity of gibberellins (GAs), leading to degradation of DELLA proteins and promotion of the shade-avoidance syndrome (SAS). In turn, activation of the JA pathway by molecular patterns associated with herbivores and some pathogens triggers JA biosynthesis, which leads to defense by increasing the 26S-mediated turnover of JAZ repressors. DELLAs and JAZs impair each other's ability to interact with their target transcription factors. Low R:FR ratios appear to shift the balance from defense to SAS responses by causing increased DELLA degradation (and hence promoting JAZ activity) and by increasing JAZ stability. Arrows indicate positive interactions; truncated connectors indicate negative regulation. Orange arrows denote protein turnover. Dashed lines denote regulation for which the biochemical mechanism is unclear. Key regulators and outcomes are highlighted with gray shadows. Additional abbreviations: DAMP, damage-associated molecular pattern; HAMP, herbivore-associated molecular pattern; PAMP, pathogen-associated molecular pattern; PIF, PHYTOCHROME-INTERACTING FACTOR; Pfr, active form of phyB; Pr, inactive form of phyB.

via increased DELLA stability, as explained above. However, the *sav3* mutant of *Arabidopsis*, which does not increase auxin biosynthesis in response to low R:FR (154), still shows depressed immunity under supplemental FR when tested in bioassays with chewing insects and necrotrophs (30, 120).

Brassinosteroids, which also participate in the orchestration of the SAS phenotype induced by low R:FR ratios in *Arabidopsis* (100), can antagonize JA-mediated growth and antiherbivore responses (27, 136) while increasing SA accumulation and *PR1* expression (138). Brassinosteroids also modulate the efficiency of plant immune responses elicited by microbe-associated molecular patterns (3, 14). Whether the increased activity of growth-promoting hormones caused by low R:FR ratios can account for the reduced antiherbivore defenses at high density or under canopy shade has not been explicitly studied.

Phytochrome and salicylic acid. Mutants of *Arabidopsis* and rice carrying the *phyB* mutation (alone or in combination with mutations in other *PHY* genes) allow stronger proliferation of and/or are more susceptible to (hemi)biotrophic pathogens than the corresponding wild types (48, 59, 63, 163). A recent study used ecologically realistic manipulations of the light environment and found that plants exposed to low R:FR at the time of inoculation with the virulent hemibiotrophic pathogen *P. syringae* pv. *tomato* DC3000 were more susceptible than those exposed to high R:FR (38). Similarly, *phyB* plants supported significantly higher bacterial proliferation than the Col-0 wild type. Increased susceptibility to *P. syringae* pv. *tomato* DC3000 correlated with reduced expression of the marker gene *PR1*, suggesting impaired SA signaling under low R:FR. Interestingly, SA treatment did not repress the responses of the SAS marker gene *PIL1* to low R:FR ratios (38). Thus, the effect of phyB inactivation reducing plant resistance to biotrophic pathogens is analogous to that reported for necrotrophs and chewing insects, and is consistent with a hierarchy in which, at least at the level of transcription, SAS responses have priority over defense responses when plants are simultaneously exposed to signals of competition and pathogen attack.

Mechanisms of phytochrome B–salicylic acid crosstalk. Little is known about the mechanisms underlying the effects of shade-light signals on plant defenses against biotrophs. Analysis of microarray data indicated that low R:FR ratios cause a massive decrease in the expression of SA-induced genes, and most of the genes that were downregulated by low R:FR are known to be NPR1 dependent (38). Intriguingly, low R:FR ratios appear to promote nuclear localization of NPR1, perhaps as a consequence of changes in the redox state of the cell caused by uneven excitation of photosystems I and II under FR-enriched light conditions. Increased nuclear import of NPR1 would be expected to enhance, rather than inhibit, SA responses (122). Genes coding for several SA-induced kinases were inhibited by low R:FR ratios, and indeed NPR1 phosphorylation did not increase proportionally to the increase in NPR1 monomers under low R:FR (38). Because phosphorylation of NPR1 monomers and subsequent proteasome-mediated turnover are essential for full expression of SA-induced transcription (150), it seems likely that the effect of simulated shade light repressing SA responses could be mediated by repression of phosphorylation cascades (38) (**Figure 3*a***).

Of course, changes in the levels of other plant hormones, brought about by phyB inactivation, could also affect SA signaling, as discussed above. In this regard, it is important to note that increased GA activity (and DELLA degradation) under low R:FR ratios—which, also as discussed above, might provide a simple model to explain negative effects of shading and competition on JA signaling—cannot explain reduced SA responses, as GAs are known to be positive regulators of SA signaling (124, 138).

UVR8 and Solar UVB Radiation

UVB radiation (280–315 nm) is a very small fraction of the solar spectrum (<1% of the quanta between 290 and 700 nm), and it is significantly attenuated as it passes through a plant canopy (61). Exposure to solar UVB radiation typically increases plant resistance to a variety of consumer organisms (9, 10, 101).

Evidence from field studies. More than 80% of the studies that have evaluated the effects of ambient UVB on insect herbivory reported that plant damage or insect growth increased in response to attenuation of UVB radiation (10). Insect-feeding experiments with plants pretreated with either ambient or attenuated UVB radiation have clearly demonstrated that at least part of the effect of solar UVB reducing herbivory is indirect (i.e., mediated by changes in the quality

of plant tissues) (28, 112, 171). More limited evidence also indicates that ambient levels of UVB can reduce infection by necrotrophs (40, 64), and experiments in which plants were pretreated with different UVB doses before inoculation with the pathogen demonstrated that natural levels of UVB can increase plant resistance to infection (40).

The effects of solar UVB radiation increasing plant resistance to herbivores and pathogens have been linked to UVB-induced changes in secondary metabolites, including leaf phenolics (40, 142), diterpenes (43), and, in some cases, defense-related proteins such as proteinase inhibitors (PIs) (39, 81, 152). The effects of solar UVB on defensive chemistry can be considered specific photomorphogenic effects, presumably mediated by specific UVB photoreceptors. In fact, strong effects of solar UVB radiation on secondary chemistry were observed under conditions in which (*a*) UVB caused negligible inhibition of photosynthesis and (*b*) the role of other photoreceptors can be excluded because the UVB irradiances were very low relative to the irradiances at the principal wavelengths absorbed by phytochromes or B-light photoreceptors (13, 110). In addition, in more recent studies with *Arabidopsis*, the effects of solar UVB (or physiologically meaningful doses of UVB radiation) increasing the accumulation of flavonoids and other soluble phenolic compounds depended on UVR8 (40).

UVB and the jasmonate–salicylic acid backbone. In spite of the well-documented effects of UVB on plant defense, the connections between UVB radiation and the backbone of defense signaling (i.e., the JA and SA pathways) are much less well characterized than those described between these defense pathways and phyB (**Figure 3*b***). Early reports of UV effects on SA and expression of SA marker genes (such as *PR1*) (164) should be interpreted cautiously, as many of those experiments used UV doses (or wavelengths) not present in the terrestrial environment (such as UVC, <280 nm), or unbalanced UVB treatments (high UVB delivered against low PAR levels). Similar limitations apply to early studies of UV effects on JA activity (36).

Because the best-characterized effects of UVB on defense come from experiments that tested plant resistance to herbivorous insects and necrotrophic pathogens, work in the past few years has focused on interactions with JA signaling (**Figure 3*b***). Evidence in *Arabidopsis* (28) and *Nicotiana* (39) indicates that genetic perturbations that affect accumulation of bioactive JAs can effectively cancel some antiherbivore effects of solar UVB radiation under field conditions. To date, these data constitute the best evidence for the involvement of JA signaling in the effects of solar UVB increasing plant resistance to herbivory.

Mixed results have been obtained regarding effects of ecologically meaningful UVB treatments on accumulation of JAs. Studies with *N. longiflora* and *N. attenuata* reported increased transcript levels of genes encoding JA biosynthesis enzymes in plants exposed to solar UVB compared with plants exposed to attenuated UVB (81). A recent study in *Arabidopsis* found UVR8-dependent transcription of several genes related to JA biosynthesis and signaling after exposure to solar UV radiation (117). Regarding JA hormone levels, Đinh et al. (43) reported that attenuation of solar UVB resulted in lower levels of JA and JA-Ile in *N. attenuata* plants on some sampling dates of a multiyear field study. In contrast, Demkura et al. (39) failed to detect higher levels of JA or JA-Ile in response to supplementation with natural UVB irradiances in a greenhouse study with *N. attenuata*. It would seem that the effects of natural UVB on JA levels are subtle and variable with time and the occurrence of other environmental stressors. Part of the variation might result from buffering effects of UVB-induced flavonoids on JA signaling (see UVB and Leaf Phenolics, below).

Besides JA synthesis, effects of UVB radiation increasing plant sensitivity to JA have been documented, particularly in experiments with solanaceous species that quantified PI activity or *PI* gene expression (39, 81, 152). In contrast, experiments with *Arabidopsis* failed to detect a significant

effect of realistic UVB treatments on plant sensitivity to JA (40). Clearly, more studies are needed to understand how solar UVB radiation interacts with JA and SA signaling under field conditions.

UVB and leaf phenolics. UVB radiation can affect plant defense against herbivores and pathogens via mechanisms that are not mediated by JA (**Figure 3*b***). For example, UVB radiation, acting through UVR8, increases the resistance of *Arabidopsis* plants to *B. cinerea*, and this effect is conserved in JA-insensitive genotypes, such as *jar1* and a transgenic line that overexpresses a stable JAZ10 splice variant (JAZ10.4) (40). It is well established that UVB radiation promotes the accumulation of certain phenolic compounds in a JA-independent manner, including flavonoids (C15 molecules) (39, 94) and C3-C6 phenylpropanoids (40). Flavonoids, such as rutin, can have an antiherbivore function (45, 70, 151). In addition, according to recent evidence, increased flux through the flavonoid pathway can result in reduced JA and JA-Ile accumulation responses to wounding in *Arabidopsis* (131). This effect of flavonoids, as buffers of JA responses, may be one reason that some studies found reduced levels of herbivore-induced JA and JA-Ile levels in plants exposed to flavonoid-inducing UVB treatments (39) and could explain some of the variability among experiments regarding UVB effects on levels of bioactive JAs (as discussed in UVB and the Jasmonate–Salicylic Acid Backbone, above).

Regarding C3-C6 phenylpropanoids, recent work in *Arabidopsis* suggests that the effect of UVB increasing plant resistance to *B. cinerea* is mediated by the UVR8 photoreceptor via stimulation of sinapate biosynthesis (40). Sinapates can serve as precursors in the synthesis of syringyl lignin, which plays a role in cell wall fortification and pathogen resistance (98, 106).

Cryptochromes

In contrast to the well-documented effects of R:FR ratio and solar UVB on plant immunity, evidence for an important role of specific B-light signals is scant (10). A positive effect of cry1 on *Arabidopsis* resistance to *P. syringae* pv. *tomato* DC3000 was reported for plants exposed to continuous light after pathogen infection (161). However, other studies, carried out under day/night light cycles and using a range of microbial pathogens, did not find evidence for specific effects of cryptochrome photoreceptors on plant defense (30, 63, 84).

PHOTOSYNTHESIS AND PLANT DEFENSE

In addition to activating specific photoreceptors, it is obvious that a main effect of light on plants is to stimulate CO_2 uptake through stomata and provide energy for CO_2 fixation through photosynthesis. Whereas the effects of variation in light level and chloroplast-derived signals on immune responses have been documented in several pathosystems (88, 90), there is surprisingly limited evidence indicating that the variations in photosynthetic activity that take place in plant canopies are actually responsible for the variations in plant sensitivity to insects or pathogens associated with different levels of shading or population densities. Kangasjärvi et al. (88) recently reviewed the literature addressing the role of photosynthesis as a modulator of the energy status of the cell (which affects its ability to mount a defense response) and in the generation of reactive oxygen species with potential connections with defense signaling. I refer readers to this comprehensive review for an in-depth treatment of the subject.

Changes in PAR levels could affect plant resistance to pathogens and herbivores through indirect mechanisms. For example, changes in photosystem II excitation pressure, resulting from irradiance changes, can affect leaf morphology (78). High PAR often results in thicker leaves (18), and increased leaf thickness may provide resistance against chewing insects (144). Obviously, changes

in PAR levels also affect production of soluble sugars, and sugars can activate defense-related responses (16) and modulate key signaling elements of defense hormone pathways (168). Finally, for plants growing under natural conditions, PAR-induced changes in stomatal conductance, which are to a large extent mediated by photosynthetic activity, can have important implications for the distribution of xylem-transported CKs. Leaves that are more exposed to sunlight attract more transpiration water than shaded leaves and therefore have higher levels of CKs (20). CKs are positive modulators of JA synthesis (41) and SA responses (32).

CONCLUDING REMARKS AND THE VALUE OF ECOPHYSIOLOGY

Adjusting the Balance Between Competitive Ability and Defense

The evidence reviewed here demonstrates that, for shade-intolerant species, if the risk of competition sensed by photoreceptors is high, then expression of the SAS phenotype takes top priority, while plant defenses are largely suppressed. From an adaptive perspective, the priority on elongation and related SAS traits can be explained on the basis of the high cost associated with losing the race for the capture of PAR in rapidly growing canopies. Suppression of defense responses presumably saves resources for growth or helps the plant focus its defense effort on its most valuable organs (see below). The main mechanism for reduced expression of defense under competition appears to be the simultaneous suppression of JA and SA signaling by low R:FR ratios, although attenuation of UVB-induced defenses may also play a significant role under field conditions. This effect of light signals perceived by photoreceptors on the JA-SA backbone of plant immunity provides a mechanistic explanation for the long-established patterns of reduced plant defenses under conditions of high density and leaf shading.

Signal Crosstalk and Ecophysiology

To understand the ecophysiology and adaptive value of the photomodulation of defense signaling, it is important to scale up to a whole-plant perspective. This implies consideration of how the defense effort is distributed through the whole plant and how environmental conditions may affect within-plant signaling.

Defense is regulated at the modular level. For obvious reasons, most studies on plant immunity have been carried out using whole *Arabidopsis* seedlings. This is a great system to study signal crosstalk, but it does not help as much in understanding how the plant's defense strategy is distributed among different plant parts. Plants are constructed by reiteration of basic modules (phytomeres), and this is a central aspect of their developmental strategy. Unlike most animals, plants "sacrifice" certain organs or parts when they are not useful (for example, shaded leaves) and grow new ones if doing so is convenient for resource capture.

Modularity also helps plants distribute the defense effort according to the relative value of the various phytomeres to be defended. It has long been recognized that young leaves are better defended against herbivores and pathogens than old ones (114). This within-plant distribution of defenses makes functional sense, as young leaves concentrate most of the plant's nitrogen and are major contributors to CO_2 uptake. From a mechanistic point of view, however, an important question is how the value of a leaf (or other organ) is established at the molecular level. In some cases, value may be defined by ontogeny, and hormonal profiles associated with different developmental stages (e.g., leaf primordium versus mature leaf) may help tailor the expression of the plant immune system accordingly (115). However, in many cases, particularly for plants grown

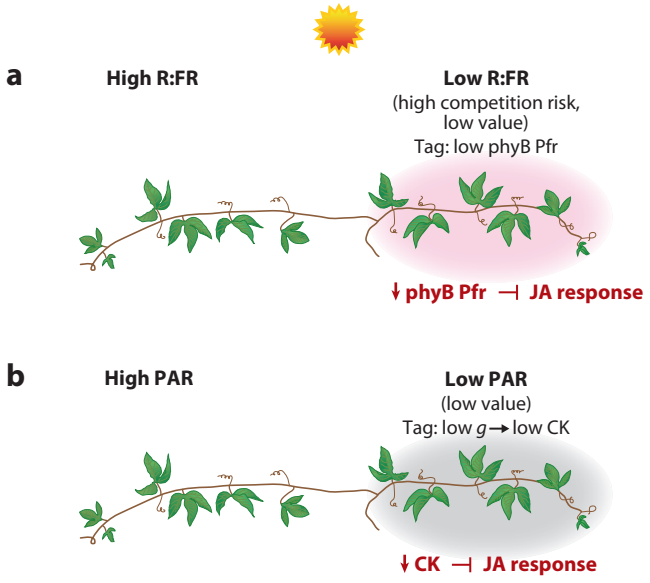


Figure 5

How light defines leaf value and investment in defense: two mechanisms by which plants can concentrate defenses in well-illuminated, photosynthetically valuable branches. (*a*) Branches receiving low red:far-red (R:FR) ratios, even at high levels of photosynthetically active radiation (PAR), are likely to face stronger competition for light in the long run than those receiving high R:FR. Empirical evidence in passion fruit indicates that defense responses in these "doomed-to-fail" branches are repressed, and this repression is mediated by a reduction in sensitivity to jasmonate (JA) caused by phytochrome B (phyB) inactivation (79). (*b*) Shaded leaves have lower stomatal conductance (*g*) and reduced transpiration rates compared with leaves exposed to high PAR. This creates a gradient in cytokinin (CK) concentrations, which are higher in well-illuminated leaves, and a potential mechanism to tailor defense expression to the photosynthetic value of each leaf based on the positive effect of CK on JA and salicylic acid signaling. Additional abbreviation: Pfr, active form of phyB.

in patchy and dynamic canopy environments, leaf value is more likely to be defined as a positive function of light exposure.

Signals generated by phyB (and presumably other photoreceptors, such as UVR8) can help concentrate the defensive arsenal of each plant on leaves that meet with the best light conditions (**Figure 5*a***). For example, it has been shown recently that phyB can locally modulate JA responses. Studies using the vine *Passiflora edulis* (79) showed that the production of extrafloral nectar (an indirect defense) can be induced by herbivory and JA treatment and that this induction is localized (i.e., restricted to the branches that were exposed to the induction treatment). Furthermore, the local defense response was locally suppressed if the induced branch was simultaneously exposed to light with a low R:FR ratio.

These observations do not necessarily contradict the well-established concept of plant defense theory that induced defenses are systemically expressed. Systemic induction was a key result in the pioneering experiments of Green & Ryan (62) and was subsequently demonstrated for JA and SA responses. However, in plants that are more complex than a seedling, there appears to be room for considerable variation among organs in the degree of expression of induced defenses. The results with *P. edulis* suggest that the value of a branch or module is determined by the level of phyB Pfr, and that local suppression of JA signaling by a low R:FR ratio is a mechanism that concentrates defense resources in well-illuminated parts of the plant. When plants grow in a real canopy, only

leaves exposed to the lowest R:FR ratios would be expected to experience defense attenuation; these leaves are likely to be sacrificed in order to focus plant resources on apical growth (SAS) and protection of leaves placed in more favorable light conditions.

Light-induced hormonal changes can vary with environment and among organs. Another mechanism to explain the concentration of defense in leaves with high photosynthetic activity is based on the hypothesis that CKs, which are distributed following gradients of transpiration intensity (and thus exposure to sunlight), act as a molecular tag for leaf value (8) (**Figure 5*b***). For simplicity, most studies of hormonal regulation of plant immunity use seedlings grown under conditions of minimal transpiration and photosynthesis (closed Petri dishes, liquid media often containing sucrose, etc.). Clearly, to fully understand the role of PAR-induced hormonal profiles in plant immunity, it is imperative to conduct studies under ecologically meaningful PAR levels and under conditions in which plants are exposed to realistic evaporative demands. In the same vein, the majority of studies on phyB regulation of hormone changes used very young *Arabidopsis* seedlings as a model system (72, 96, 105, 154) and, for example, analyzed auxin transport to the hypocotyls in great detail to explain changes in hypocotyl elongation (96). Similarly, detailed studies of increased GA activity (103) or DELLA turnover (44, 95) triggered by competition signals have focused on hypocotyls or rapidly elongating petioles. Changes in the levels of auxin and GA are assumed to modulate JA and SA responses. However, it remains to be tested whether low R:FR ratios induce sufficient changes in levels of growth-related hormones in the leaf laminas of more mature plants, which are the plant parts most often used to test for regulation of defense responses and plant susceptibility to pathogens and insects.

Agronomy and Biotechnology

Increased demand for food and other commodities of agricultural origin will continue to put pressure on agricultural systems. Current estimates by the Food and Agriculture Organization of the United Nations indicate that, to cope with this demand, agricultural production would need to increase by 70% by 2050, and 90% of the growth in crop production would have to result from higher yields and increased cropping intensity (24). Because of the need for increasing yields per unit area, the use of high planting densities and technology that increases LAI early in the season to maximize the interception of solar radiation has become mandatory. As demonstrated by the evidence discussed in this review, increased plant density would imply additional problems in the control of pests and diseases. A recent analysis suggested that despite the massive increase in the use of pesticides during the past four decades, the overall proportion of crop losses to pests and diseases has not decreased (125). In addition, chemical control strategies are facing growing public rejection and are becoming increasingly regulated owing to their negative impacts on human health and ecosystems (17). The major recent advances in descriptions of the mechanisms of plant immunity and their regulation by canopy light can provide the basic elements to design crop plants that are less dependent on the use of synthetic pesticides and maintain a robust defense system over a wide range of planting densities.

SUMMARY POINTS

1. Precise allocation of limited resources to improve competitive ability and defense is critical for plant survival, and conditions of high competition for light are often associated with increased susceptibility to pathogens and herbivores.

2. Perception of competition signals by informational photoreceptors suppresses the expression of defense responses to pathogens and insects.
3. The jasmonate (JA)–salicylic acid (SA) hormonal backbone provides a conceptual framework to study the regulatory roles of internal and external signals on plant defense.
4. The main mechanism for reduced expression of defense under competition appears to be the simultaneous suppression of JA and SA signaling by low red:far-red ratios.
5. Inactivation of phytochrome B (phyB) by competition signals appears to suppress JA responses by altering the balance between DELLA and JAZ proteins in favor of the latter.
6. Solar UVB radiation is a positive modulator of plant defense, acting through mechanisms that involve JA-dependent and JA-independent pathways.
7. Light, acting through phyB and presumably other mechanisms, defines the value of different leaves and helps plants to concentrate their defensive arsenals in modules that meet favorable light conditions.

DISCLOSURE STATEMENT

The author is not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

I thank Amy Austin, Carlos Mazza, Javier Moreno, Ronald Pierik, and Johannes Stratmann for their critical reading of the manuscript and many helpful suggestions. Work in my laboratory is supported by grants from CONICET, UBACyT, and the Agencia Nacional de Promoción Científica y Tecnológica.

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Annual Review of Plant Biology
Volume 65, 2014

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Errata

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