

Canopy Light and Plant Health¹

Carlos L. Ballaré*, Carlos A. Mazza, Amy T. Austin, and Ronald Pierik

Instituto de Investigaciones Fisiológicas y Ecológicas Vinculadas a la Agricultura, Consejo Nacional de Investigaciones Científicas y Técnicas, Universidad de Buenos Aires, C1417DSE Buenos Aires, Argentina (C.L.B., C.A.M., A.T.A.); and Plant Ecophysiology, Institute of Environmental Biology, Utrecht University, 3584 CH Utrecht, The Netherlands (R.P.)

During the course of this century, increasing human population and economic development will continue to put pressure on agricultural systems for increased crop yields (Rosegrant and Cline, 2003; Foley et al., 2005). Grains produced for livestock feed and biofuels are now competing for space with crops intended for human consumption (Foley et al., 2011), which means that in order to meet increasing global food demand and multiple objectives for arable land, new crop varieties with improved performance will have to be developed. At the same time, the use of fertilization, irrigation, and pesticides is very likely to increase (Oerke, 2006; Jaggard et al., 2010). The intensification of agricultural practices, however, creates environmental and health concerns, which are already a matter of significant scientific and social debate. Alternative methods to increase crop yields without agricultural expansion and under conditions of reduced chemical use are urgently required.

A particular concern for the growing intensity of agricultural practices is the management of pests and diseases, which can account for up to 25% of preharvest crop losses in agricultural areas managed with well-developed crop protection technologies (Pimentel, 1997; Oerke, 2006). In order to minimize these losses, expensive and potentially hazardous chemical control strategies are routinely applied, which are becoming increasingly regulated due to their negative impacts on human health and ecosystems (Birch et al., 2011). Furthermore, a recent analysis suggests that despite the massive (greater than 15-fold) increase in the use of pesticides during the past four decades, the overall proportion of crop losses to pests and diseases has not decreased (Oerke, 2006). It is interesting that while these levels of crop losses in agroecosystems are maintained at a high cost, in natural ecosystems plant biomass losses due to insect herbivory are typically fairly small (approximately 10%; Schoonhoven et al., 2005). It seems possible that lessons could be learned

from natural defense mechanisms to reduce negative impacts of pests and pathogens and, consequently, the inputs of toxic chemicals used to protect crops and crop yield from biotic stressors. Indeed, plants have demonstrated a broad repertoire of extremely effective tactics that allow them to fight off natural enemies (Karban and Baldwin, 1997; Schoonhoven et al., 2005; Jones and Dangl, 2006; Wu and Baldwin, 2010; Agrawal and Heil, 2012).

The mechanisms responsible for plant defense are becoming increasingly well understood at the molecular level (Jones and Dangl, 2006; Howe and Jander, 2008; Panstruga et al., 2009; Wu and Baldwin, 2010). Major hormonal players in the regulation of immune responses have been identified (Erb et al., 2012; Pieterse et al., 2012), and the functions of defense-related molecules produced by plants are gradually becoming elucidated (D'Auria and Gershenzon, 2005; Mithöfer and Boland, 2012). Our understanding of the regulation of defense systems by environmental signals or conditions is also increasing rapidly, and the discipline of plant biology is creating the knowledge base and conceptual foundation for the purposeful utilization of natural defense mechanisms in agriculture.

Light has emerged as a key modulator of plant immunity. However, until very recently, the beneficial effects of light on plant resistance to pests and disease have been largely unappreciated. In this Update, we briefly highlight recent findings in this area, focusing on how changes in the canopy light environment, caused by the proximity of other plants, regulate plant immunity. We propose that a better understanding of the underlying mechanisms is essential for the development of healthier crop systems necessary for reducing the environmental costs of modern agriculture.

PROXIMITY EFFECTS

A major imperative for modern agriculture is to increase crop yield per unit area, due to the escalating demand of agricultural commodities and the simultaneous need for preservation of natural ecosystems. A common strategy in the management of many crops for enhancing yield has been the implementation of practices that increase canopy light interception, such as higher planting density, reduced row spacing, and fertilization (Harder et al., 2007; De Bruin and Pedersen,

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* Corresponding author; e-mail ballare@ifeva.edu.ar.
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2008). All of these practices, which result in increased leaf area index (LAI), can substantially reduce individual plant exposure to solar radiation, particularly early in the growing season (Flénet et al., 1996; Maddonni et al., 2001).

The effects of plant density on the severity of plant disease caused by microbial pathogens are well documented in natural and managed ecosystems (Burdon and Chilvers, 1975; Augspurger and Kelly, 1984; Bell et al., 2006; Jurke and Fernando, 2008), with a general pattern of increased disease at high plant densities (for review, see Burdon and Chilvers, 1982; Alexander and Holt, 1998; Gilbert, 2002). Similarly, high density and shading frequently increase plant damage by herbivorous insects and/or reduce the expression of antiherbivore defenses (Karban et al., 1989; Cipollini and Bergelson, 2001, 2002; Yamamura, 2002; Roberts and Paul, 2006; Agrawal et al., 2012). The mechanisms that mediate these effects of crowding on plant health are elusive. In the case of microbial pathogens, micro-environmental factors such as air humidity and leaf surface wetness are likely to play an important role. 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 (Roberts and Paul, 2006). In the following sections, we will discuss how changes in the canopy light environment, caused by increased LAI and perceived by specific photoreceptors, affect the expression of plant defenses against biotic stressors.

UV-B AND UVR8

Overcrowding, air pollution, and poorly lit urban environments are widely accepted to have played a role in the increased incidence of rickets, a bone disorder of childhood, during the early days of the industrial revolution in the 19th century (Holick, 2004). A link between sunlight deprivation and rickets was originally postulated in the early 1800s and experimentally confirmed nearly a century later by studies showing that the disease could be cured by exposing children with rickets to the radiation produced by a mercury arc lamp. These observations led, perhaps, to the first appreciation of the beneficial effect of sunlight and UV radiation on human health, an effect that is now documented and mechanistically linked with the photosynthesis of vitamin D driven by UV-B radiation (280–315 nm; Holick, 2004, 2007; Juzeniene et al., 2011).

Positive effects of UV-B radiation on plant health have also been demonstrated, in spite of the fact that the vast majority of the studies of UV-B impacts were originally aimed to detect deleterious effects of UV-B on plant growth. Most examples of “beneficial” effects of UV-B on plants come from studies of plant-herbivore interactions. Field experiments in which losses to phytophagous insects were compared between plants grown under either ambient or attenuated levels of

solar UV-B radiation have shown that plant exposure to solar UV-B radiation typically increases plant resistance to insect herbivory (for review, see Caldwell et al., 2003; Ballaré et al., 2011; Kuhlmann and Müller, 2011; Fig. 1). UV-B radiation has also been reported to increase plant resistance to microbial pathogens (Gunasekera et al., 1997; Wargent et al., 2006; Gunasekera and Paul, 2007; Kunz et al., 2008; Demkura and Ballaré, 2012) and can interact with the plant to modify the composition of microbial communities in the phyllosphere (Kadivar and Stapleton, 2003; Balint-Kurti et al., 2010). Treatment with unnaturally high doses of UV-B radiation, or with UV wavelengths not present in the daylight spectrum (UV-C, $\lambda < 280$ nm), has also been reported to activate defense-related pathways in several species (Bridge and Klarman, 1973; Brederode et al., 1991; Conconi et al., 1996; Mert-Turk et al., 2003; Glawischnig, 2007); however, the ecological significance of these responses is not clear.

The effect of natural levels of UV-B radiation boosting plant resistance to biotic stress has been linked with

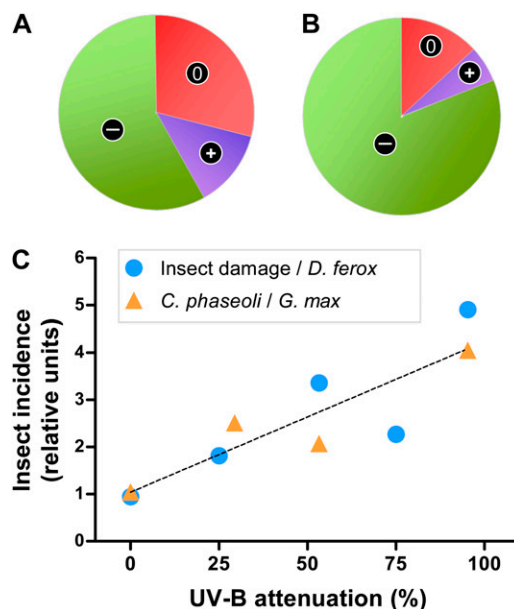


Figure 1. Solar UV-B radiation increases plant resistance to herbivorous insects in the field. A, Fraction of published studies showing that UV-B radiation reduced (–), increased (+), or did not affect (0) herbivory levels or insect performance (adapted from Kuhlmann and Müller, 2011). B, Same as in A, but analysis restricted to studies that tested the effects of solar (ambient) UV-B radiation (i.e. excluding UV-B lamp supplementation studies). C, Quantitative relationship between solar UV-B attenuation and insect herbivory under field conditions. UV-B attenuation, which was accomplished with plastic films that reduced the UV-B component of solar radiation with minimal effects on other wavelengths, increased the density of insect herbivores (*Caliothrips phaseoli*) in a soybean canopy and damage caused by leaf beetles on *Datura ferox* plants. Zero percent attenuation corresponds to full sunlight. Primary data can be found in Ballaré et al. (1996) and Mazza et al. (1999).

increased expression of jasmonate (JA) responses and with the induction of increased levels of phenolic compounds. Indirect evidence for a connection with JA responses originated from the observation that plant exposure to solar UV-B can increase the expression of JA-responsive markers, such as proteinase inhibitors, in tomato (*Solanum lycopersicum*) and *Nicotiana* spp. (Stratmann et al., 2000; Izaguirre et al., 2003; Demkura et al., 2010). However, synergistic effects of UV-B and JA elicitation have not been observed for several classic markers of the JA response in *Arabidopsis* (*Arabidopsis thaliana*; Demkura and Ballaré, 2012). Similarly, mixed results have been obtained regarding the effects of UV-B radiation on JA accumulation (A-H-Mackerness et al., 1999; Demkura et al., 2010). The strongest evidence for a role of JA in some defense responses triggered by natural doses of UV-B radiation comes from studies using mutants or transgenic lines impaired in JA biosynthesis or signaling. Disruption of the JA signaling pathway in *Arabidopsis* (Caputo et al., 2006) and *Nicotiana attenuata* (Demkura et al., 2010) was shown to eliminate the effect of solar UV-B inducing plant protection against herbivorous insects. In contrast, a recent case was reported in which a positive effect of UV-B radiation on plant resistance against pathogens was conserved in JA-response mutants (Demkura and Ballaré, 2012), suggesting that UV-B radiation likely influences plant defense via multiple pathways.

Soluble phenolic compounds typically accumulate in plants exposed to solar UV-B radiation and play a central role in photoprotection (Caldwell et al., 1983; Mazza et al., 2000; Kotilainen et al., 2009). Interestingly, it has been noted that some of the phenolic compounds that are induced by UV-B exposure are also induced by insect herbivory and other biotic stressors (Izaguirre et al., 2007; Demkura et al., 2010). The partial convergence in the induction of phenolic metabolites, and the fact that some of these compounds may have a role as defenses against pests and pathogens, have lent support to the idea that at least part of the effect of UV-B radiation on plant resistance to biotic stressors is mediated by the enhanced production of phenylpropanoid derivatives. For example, enhancement of JA- and insect-induced phenolic responses by solar UV-B radiation has been documented for conjugated polyamines in *N. attenuata* (Demkura et al., 2010). Recent studies in this species have identified MYB8 as a critical transcription factor controlling polyamine synthesis in response to herbivory (Onkokesung et al., 2012), and the MYB8 gene is known to be positively regulated at the transcriptional level by UV-B radiation (Pandey and Baldwin, 2008). It is worth noting that stress-specific alterations in phenolic profiles have also been reported (Demkura et al., 2010; Kuhlmann and Müller, 2011): for example, flavonoids and sinapates typically accumulate in response to UV-B irradiation but not in response to herbivory or JA treatments (Demkura et al., 2010; Demkura and Ballaré, 2012).

The mechanism of perception of UV-B radiation is becoming increasingly elucidated (Jenkins, 2009; Heijde and Ulm, 2012), and a UV-B photoreceptor, UV-RESISTANCE LOCUS8 (UVR8), has been recently characterized at the molecular level (Rizzini et al., 2011). UVR8 is a β -propeller protein that under UV-B-free light conditions exists as a homodimer. Absorption of UV-B quanta by UVR8 induces instant monomerization of the photoreceptor, its accumulation in the nucleus, and interaction with the E3 ubiquitin ligase CONSTITUTIVELY PHOTOMORPHOGENIC1 (Rizzini et al., 2011), which is required for UV-B-stimulated gene activation in light-grown seedlings (Oravec et al., 2006). This increased understanding of UV-B perception will greatly facilitate research on the mechanism by which UV-B radiation affects the expression of plant immune responses.

Whereas the role of UVR8 in UV-B-induced gene expression is now well established (Jenkins, 2009; Heijde and Ulm, 2012), the participation of this photoreceptor in the activation of plant defenses against biotic stress requires further study. Conceivably, the effects of UV-B on plant defense might also result from pleiotropic consequences of UV-induced cellular damage and activation of a "generalized" stress response (Brown and Jenkins, 2008; González Besteiro et al., 2011). Nevertheless, it is important to note that, under field conditions, strong impacts on plant defense and health are induced by relatively low UV-B irradiances (less than 1% of the total short-wave sunlight photon flux), which do not cause visible stress symptoms or plant damage (discussed in Demkura et al., 2010). Therefore, a specific, photoreceptor-activated signaling pathway is likely to be involved in the regulation of plant immunity by UV-B radiation.

Recently, activation of UVR8 was linked to the production of chemicals involved in plant defense against pathogens (Demkura and Ballaré, 2012; Fig. 2). Small, ecologically meaningful doses of UV-B radiation (approximately $1 \mu\text{mol m}^{-2} \text{s}^{-1}$; $\lambda > 290 \text{ nm}$) were shown to increase the resistance of *Arabidopsis* plants to the necrotrophic fungus *Botrytis cinerea* in a UVR8-dependent manner. The same study showed that UVR8 was required for UV-B-induced accumulation of sinapates (sinapoyl malate and sinapoyl Glc) and that the UV-B effect on fungal resistance was absent in *fah1*, a mutant deficient in ferulic acid 5-hydroxylase, which is essential for sinapate biosynthesis. Sinapates are important precursors in the synthesis of syringyl-type ("defense") lignin, which is used to strengthen the plant cell walls and prevent penetration by fungal hyphae (Lloyd et al., 2011). Collectively, these results indicate that UVR8 plays an important role in mediating the effects of solar UV-B radiation on pathogen resistance by controlling the expression of the sinapate biosynthetic pathway. The UV-B effect on plant resistance to *B. cinerea* was conserved in *transparent testa4* (deficient in chalcone synthase), which suggests that this effect is likely independent of UV-B-induced flavonoid accumulation (Demkura and Ballaré, 2012). However, it is important to note that flavonoid

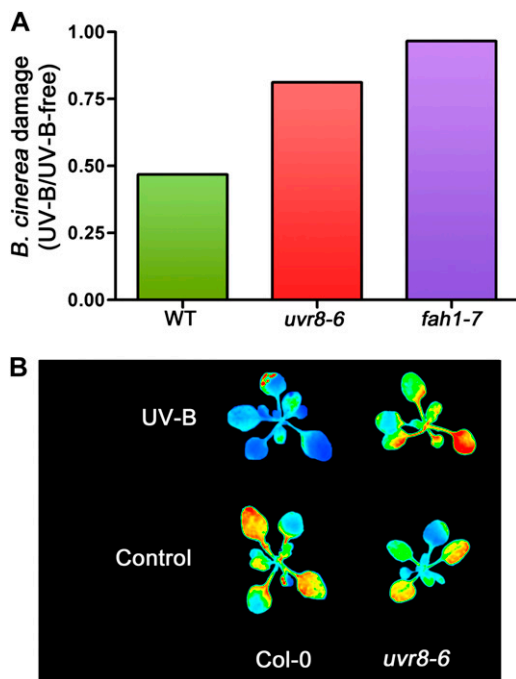


Figure 2. UV-B radiation increases Arabidopsis resistance to the necrotrophic pathogen *B. cinerea*; this effect is mediated by UVR8 via increases in sinapate accumulation. Original data can be found in Demkura and Ballaré (2012). A, Damage by *B. cinerea* in plants grown with exposure to UV-B radiation relative to damage in control plants grown under UV-B-free conditions for wild type (WT) ecotype Columbia (Col-0) plants and mutants deficient in UV-B perception (*uvr8-6*; Favory et al., 2009) and sinapate biosynthesis (*fah1-7*; Meyer et al., 1996). B, UV-B induces the accumulation of phenolic sunscreens (mostly flavonoids and sinapates) in a UVR8-dependent manner. Sunscreen accumulation is revealed here by a decrease in the intensity of UV-induced chlorophyll fluorescence. Blue color indicates higher accumulation of phenolic compounds.

biosynthesis is also controlled by UVR8 (Jenkins, 2009) and that flavonoids can play a role in plant defense against insect herbivores (Hoffmann-Campo et al., 2001). Studies under artificial laboratory conditions show that cellular responses triggered by microbe-associated molecular patterns (such as the flagellin-derived elicitor peptide flg22) can depress flavonoid responses triggered by UV-B radiation, presumably to save phenylpropanoid precursors for the synthesis of lignin and other antimicrobial compounds (Schenke et al., 2011; Serrano et al., 2012). If corroborated in the field, these studies suggest the potential for significant interactions between different biotic stressors (e.g. pathogens and herbivores) mediated by the regulation of plant responses to UV-B radiation.

The UVR8 pathway has not been directly linked with the regulation of any of the principal hormonal pathways involved in plant defense (i.e. JA and salicylate [SA]) as of yet. Demkura and Ballaré (2012) showed that the UVR8 effect on Arabidopsis resistance to *B. cinerea* was independent of JA signaling, but because interactions between natural levels of UV-B

radiation and JA-induced responses have been reported in other studies (see above), further work is needed to establish the influence of UVR8 activation on the regulation of defense signaling pathways.

As discussed previously, increased crop density has strong effects reducing the penetration of sunlight (including UV-B) into the canopy. The extent to which such a reduction in UV-B radiation affects plant defense needs to be quantified. All studies to date that manipulated UV-B levels have used filters placed above the canopy, which do not strictly mimic the effects of neighboring plants on the UV-B fluxes received by different plant organs. In spite of these limitations, recent UV-B exclusion experiments carried out with soybean (*Glycine max*) crops demonstrated that the well-documented negative effect of solar UV-B radiation on crop yield was reversed when soybean pests were not controlled by pesticide applications (Mazza et al., 2012). This observation suggests that natural levels of UV-B radiation may have a significant effect protecting crop plants from insect pests under field conditions, presumably through mechanisms involving UV-B-induced enhancement of plant defenses. The potential value of capitalizing on plants' natural responses to increased defenses with exposure to solar UV-B radiation should not be underestimated and could serve as a starting point for strategic crop improvement for maximizing these natural defense mechanisms. Moreover, some of the compounds that are induced by ambient levels of UV-B radiation, such as phenolic compounds (Mazza et al., 2000; Wargent et al., 2006; Berli et al., 2008) and antioxidants (Giordano et al., 2004), may have nutraceutical and organoleptic value (Jansen et al., 2008; Schreiner et al., 2012). Therefore, in horticultural crops, manipulation of plant responses to solar UV-B radiation could have significant implications for food quality.

RED TO FAR-RED RATIO AND PHYTOCHROME B

The red (R) to far-red (FR) ratio (R:FR) of sunlight is a critical environmental signal for plants. Low values of R:FR indicate the proximity of other plants, because plant tissues strongly absorb R photons, while FR quanta are either reflected or transmitted (Ballaré et al., 1990). In crop canopies, the R:FR of horizontally propagated radiation is directly related to the LAI of the plant stand. Plants sense the changes in R:FR using the phytochromes, particularly phytochrome B (phyB). Low R:FR values inactivate phyB by transforming the active form of the photoreceptor, Pfr, into the inactive form, Pr (Smith, 1995). Upon inactivation of phyB, basic helix-loop-helix transcription factors known as phytochrome-interacting factors accumulate in a dephosphorylated form and activate the expression of growth-promoting genes (Lorrain et al., 2008; Hornitschek et al., 2012; Li et al., 2012). This transcriptional reprogramming leads to increased production and activity of growth hormones such as auxins

and GAs, which accelerate cell expansion and promote the elongation of stems and petioles (Djakovic-Petrovic et al., 2007; Tao et al., 2008; Keuskamp et al., 2010; Hornitschek et al., 2012; Li et al., 2012). For plants, a fast rate of elongation is essential for rapid colonization of the upper canopy strata and a central element of a suite of phenotypic changes that are collectively known as the shade-avoidance syndrome (SAS; Franklin, 2008; Ballaré, 2009).

Whereas R:FR, perceived by phyB, plays a well-known role in neighbor detection and the elicitation of adaptive morphological responses (Ballaré, 1999; Dorn et al., 2000), its role as a modulator of defense expression is only beginning to be appreciated. A high R:FR (typical of open canopies) is a positive regulator of plant defense against herbivorous insects. Experiments with cucumber (*Cucumis sativus*), tomato, *N. attenuata*, and *Arabidopsis* demonstrate that inactivation of phyB, either by mutation or by exposing the plants to supplemental FR to lower the R:FR, decreases plant resistance to herbivory (McGuire and Agrawal, 2005; Izaguirre et al., 2006; Moreno et al., 2009; Fig. 3). Down-regulation of the expression of antiherbivory traits under low R:FR has been proposed to be a key element in the mechanism by which plants resolve the tradeoff between resource allocation to growth or defense (i.e. the “dilemma” of plants; Ballaré, 2009; Moreno et al., 2009).

The effect of variations of R:FR on resistance to microbial pathogens has received little attention until recently. Previous studies with a variety of horticultural species have shown that treatment of leaves with R stimulates plant resistance to a number of pathogens, compared with leaves incubated in the dark or visible light of other wavelengths (Islam et al., 1998, 2002). However, because the control for the R treatment was not established as a low-R:FR treatment, the results are

difficult to interpret in terms of plant ecology and specific phytochrome action. In fact, some of the protective effects of R in those experiments have been attributed to the stimulation of photosynthesis (Rahman et al., 2002). Very recent studies have shown that plants exposed to low R:FR values ($R:FR < 1$), which were designed to mimic realistic competition scenarios, are more susceptible to subsequent infection by necrotrophic pathogens (such as *B. cinerea*) than plants exposed to white light ($R:FR > 1$; Cerrudo et al., 2012; De Wit, 2012; Fig. 3). Similarly, *phyB* mutants of *Arabidopsis* were found to be more susceptible to necrotrophs than the corresponding wild types (Kazan and Manners, 2011; Cerrudo et al., 2012). Recent experiments demonstrate that low-R:FR treatments also reduce *Arabidopsis* resistance to the hemibiotrophic pathogen *Pseudomonas syringae* pv *tomato* DC3000 (M. De Wit and R. Pierik, unpublished data), which is consistent with the susceptibility phenotype of *phyB* mutants in *P. syringae* bioassays (Faigón-Soverna et al., 2006; Fig. 3). Moreover, in these experiments (De Wit, 2012; De Wit and Pierik, unpublished data), the effect of low R:FR depressing plant resistance to the pathogen was observed even if the change in light conditions (from high to low R:FR) was applied concomitantly with the infection treatment.

The mechanisms by which variations in R:FR regulate plant resistance to pests and pathogens have been connected with modulation effects of phyB Pfr on the signaling networks activated by the major defense hormones JA and SA.

R:FR and JA

Shading by a forest leaf canopy, which reduces both total irradiance and R:FR, can reduce JA production

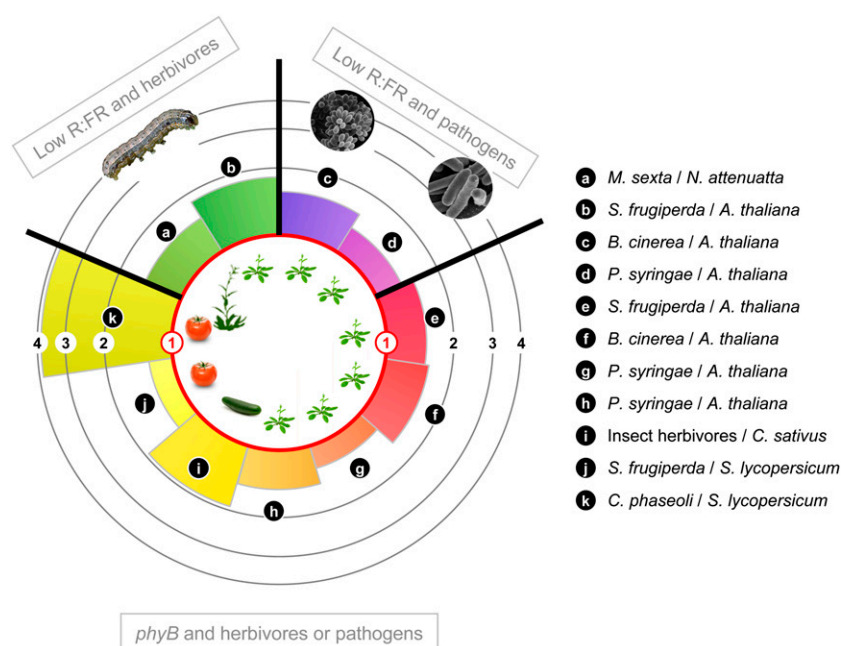


Figure 3. FR supplementation treatments, designed to mimic the proximity of neighboring plants, or mutational inactivation of the *PHYB* gene increase the levels of insect herbivory and disease severity in a variety of herbaceous species. Concentric circles indicate the level of enhancement (fold increase) of plant susceptibility to attack, expressed as a ratio between the treatment (low R:FR or *phyB*) and control (high R:FR, *PHYB*) conditions. Plant susceptibility was estimated on the basis of bioassay results that measured the growth of the consumer organism or the severity of plant damage. Sources of primary data are as follows: a, j, and k, Izaguirre et al. (2006); b and e, Moreno et al. (2009); c and f, Cerrudo et al. (2012); d and g, De Wit (2012); h, Faigón-Soverna et al. (2006); i, McGuire and Agrawal (2005).

and the expression of JA-dependent defenses in plants of the understory (Agrawal et al., 2012). Furthermore, even in the absence of shading (i.e. at constant levels of photosynthetically active radiation [PAR]), the reduction of R:FR caused by the proximity of neighboring plants in an even-height canopy can depress plant responses to JA (Moreno et al., 2009). This effect of low R:FR is mediated by the inactivation of phyB and has been demonstrated at the level of gene expression (Moreno et al., 2009; Suzuki et al., 2011; Cerrudo et al., 2012; De Wit, 2012) and the accumulation of secondary metabolites likely to be involved in direct defense against pathogens and pests, such as phenylpropanoids, anthocyanins, and glucosinolates (Izaguirre et al., 2006; Moreno et al., 2009; Cerrudo et al., 2012; Fig. 4).

The mechanisms that link phyB Pfr with JA signaling are not completely clear and are likely to involve several layers of regulation (Ballaré, 2011). Perception of jasmonoyl-Ile, the bioactive amino acid conjugate of jasmonic acid, is achieved by a coreceptor formed by the ubiquitin ligase SCF^{COI1} (for S-phase kinase-associated protein1-Cullin1-F-box protein CORONATINE INSENSITIVE1) complex and JASMONATE ZIM DOMAIN (JAZ) proteins (for review, see Browse, 2009). Jasmonoyl-Ile stimulates the specific binding of COI1 and JAZ proteins, which leads to the ubiquitination of JAZs by SCF^{COI1} and their subsequent degradation by the 26S proteasome (Chini et al., 2007; Thines et al., 2007; Yan et al., 2007, 2009; Melotto et al., 2008; Pauwels et al., 2010; Sheard et al., 2010). JAZ proteins are repressors of JA-responsive transcription factors; therefore, degradation of JAZ proteins initiates the activation of the JA response (Pauwels and Goossens, 2011; Shyu et al., 2012). Regulation of plant defense by phyB has been recently linked to JAZ activity, because the effect of low R:FR values reducing Arabidopsis resistance to *B. cinerea* was found to be significantly attenuated in a *jaz10* null mutant and in RNA interference lines disrupted for the expression of the *JAZ10* gene (Cerrudo et al., 2012). A possible mechanism behind this connection may be based on 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 values (Moreno et al., 2009). A phytochrome effect on *JAZ* stability has been demonstrated for phytochrome A (phyA). Thus, COI1-mediated degradation of *JAZ1* in response to JA treatment was found to require active phyA (Robson et al., 2010). However, it remains to be demonstrated whether in fully deetiolated plants, where responses to low R:FR are controlled predominantly by phyB (Ballaré, 1999), changes in the levels of phyB Pfr in response to R:FR affect the turnover of *JAZ* repressors.

In addition to Pfr effects on some of the early JA signaling components, light quality perceived by phyB could affect JA responses by affecting the levels of other hormones known to regulate JA signaling, and the list of these regulators includes GAs, brassinosteroids (BRs), and SA, among others (Bari and Jones,

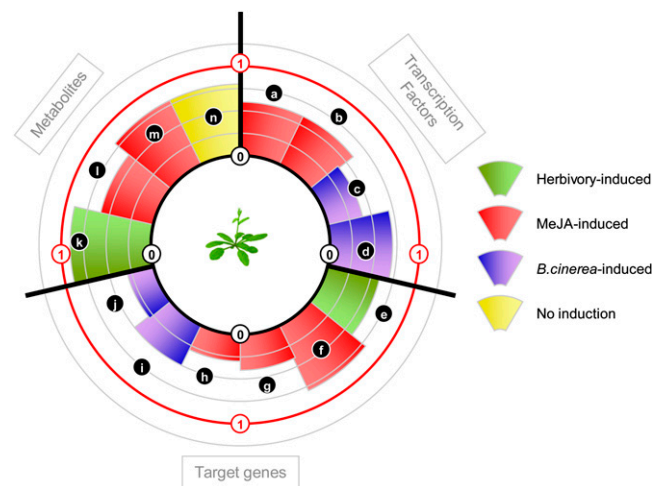


Figure 4. Exposure of Arabidopsis plants to low-R:FR treatments that mimic the proximity of neighboring plants depresses the levels of defense markers associated with JA signaling: a, *MYC2*; b, *ERF1*; c, *ERF1*; d, *ORA59*; e, *HEL*; f, *ASA1*; g, *PDF1.2*; h, *HEL*; i, *HEL*; j, *PDF1.2*; k, leaf phenolics; l, leaf phenolics; m, anthocyanins; n, glucanasturtiin. Concentric circles indicate the level of depression of the defense response caused by low R:FR, expressed as a ratio between the treatment (low R:FR or *phyB*) and control (high R:FR, *PHYB*) conditions. The elicitor used to activate the JA response is indicated by different colors. Also shown is the effect of low R:FR on the concentration of glucanasturtiin, a glucosinolate that is particularly abundant in watercress (*Nasturtium officinale*). MeJA, Methyl jasmonate. Sources of primary data are as follows: a to d, f, g, i, j, l, and m, Cerrudo et al. (2012); b, e, h, g, and k, Moreno et al. (2009); n, Engelen-Eigles et al. (2006).

2009; Ballaré, 2011; Erb et al., 2012; Pieterse et al., 2012). SA has been dismissed as a potential mediator of the effect of low R:FR values on JA signaling by studies demonstrating that this effect is conserved in SA synthesis and signaling mutants (Cerrudo et al., 2012). GAs are important growth-promoting hormones during SAS, accumulating in response to neighbor proximity and low R:FR values and participating in the elongation response (Djakovic-Petrovic et al., 2007). GAs can antagonize certain JA responses (Navarro et al., 2008), and this action is mediated by their effect on the DELLA proteins (Hou et al., 2010). GA perception leads to DELLA degradation in the proteasome (Hirano et al., 2008), and DELLAs are positive regulators of the JA response, as they prevent the repressive action of JAZ proteins on JA signaling (Hou et al., 2010). Whether the increased DELLA turnover caused by low R:FR values in canopies (Djakovic-Petrovic et al., 2007) is responsible for the depression in JA-mediated chemical defenses at high density has not been explicitly tested. BRs were recently found to participate in the orchestration of the SAS phenotype induced by low R:FR values in Arabidopsis (Kozuka et al., 2010). Interestingly, BRs have also been shown to antagonize JA-mediated growth and antiherbivore responses (Campos et al., 2009; Ren et al., 2009) and to modulate the efficiency of plant

immune responses elicited by microbe-associated molecular patterns (Albrecht et al., 2012; Belkhadir et al., 2012; Wang, 2012). BRI1-ASSOCIATED KINASE1 (BAK1) is a BR coreceptor and also physically interacts with the flg22 receptor FLS2, positively regulating FLS2-mediated innate immunity in *Arabidopsis* (Chinchilla et al., 2009). In *N. attenuata*, BAK1 is a positive regulator of herbivory-induced JA accumulation and also a negative modulator of certain defense responses elicited by JA (Yang et al., 2011). Clearly, further work is needed to assess the effects of changes in BR signaling elicited by low R:FR values on the modulation of plant defense responses in dense canopies.

R:FR and SA

SA plays a major role in disease resistance signaling (Durrant and Dong, 2004; Vlot et al., 2009). The SA response pathway is typically effective against microbial pathogens with a biotrophic lifestyle (Glazebrook, 2005). Light, when compared with dark conditions, increases plant resistance to a variety of biotrophic pathogens (Zeier et al., 2004), and SA accumulation in healthy *Arabidopsis* plants has been shown to increase in response to increased PAR (Karpinski et al., 2003). Recent experiments with cucumber suggested that light-enhanced resistance may be specific for R, since other light treatments were less effective in reducing damage by *Sphaerotheca fuliginea* (powdery mildew) as compared with R irradiation (Wang et al., 2010). Infected plants responded with increased free SA levels, and this response, together with the induction of a range of SA-responsive genes, was enhanced in R as compared with other wavelengths (Wang et al., 2010). The apparently specific effects of R suggest the involvement of phytochromes. Indeed, *phy* mutants have reduced SA-mediated resistance against (hemi) biotrophic pathogens. Thus, the *Arabidopsis phyB* mutant allows stronger proliferation of an avirulent *P. syringae* strain (Faigón-Soverna et al., 2006), and *phyAphyB* double mutants are impaired in SA-dependent systemic responses and more susceptible to pathogens with a biotrophic lifestyle (Genoud et al., 2002; Griebel and Zeier, 2008). Interestingly, defense responses at the direct sites of *P. syringae* inoculation appear to be mostly phytochrome independent (Griebel and Zeier, 2008). Triple *phyAphyBphyC* mutants of rice (*Oryza sativa*) were also shown to be more susceptible to blast fungus (*Magnaporthe grisea*) than the cv Nipponbare wild type (Xie et al., 2011). Consistent with enhanced susceptibility to this fungus, the *phyAphyBphyC* mutant showed reduced induction of *PATHOGENESIS-RELATED1* gene expression in response to SA spray treatment (Xie et al., 2011). Recent studies in *Arabidopsis* indicate that low-R:FR treatments that simulate neighbor proximity also lead to enhanced susceptibility to *P. syringae* and reduced defense gene induction in response to SA treatment (De Wit, 2012; De Wit and Pierik, unpublished data). Collectively, these results

indicate that *phyB* Pfr is a positive regulator of SA-mediated defense responses. The mechanism of SA perception has been clarified only very recently (Fu et al., 2012), and the signaling networks that connect *phyB* and SA signaling are at present largely unknown. The fact that *phyB* inactivation depresses both JA and SA responses suggests that the effect of low R:FR values on plant defense does not result from a simple shift of balance between different immune branches. Elucidation of the molecular links between light and defense signaling might reveal new connections between defense hormone pathways.

PAR, PHOTOSYNTHESIS, AND BLUE LIGHT RECEPTORS

Chloroplast-derived signals can also modulate defense responses, although a clear picture of the adaptive significance of this regulation under canopy light conditions is not yet available (Karpinski et al., 2003; Kangasjärvi et al., 2012). Similarly, the role of blue light (B) and B photoreceptors in the regulation of defense responses is not well established. Jeong et al. (2010) reported that cryptochrome 2 and phototropin 2 are specifically required for resistance protein-mediated *Arabidopsis* defense against *Turnip crinkle virus*. There

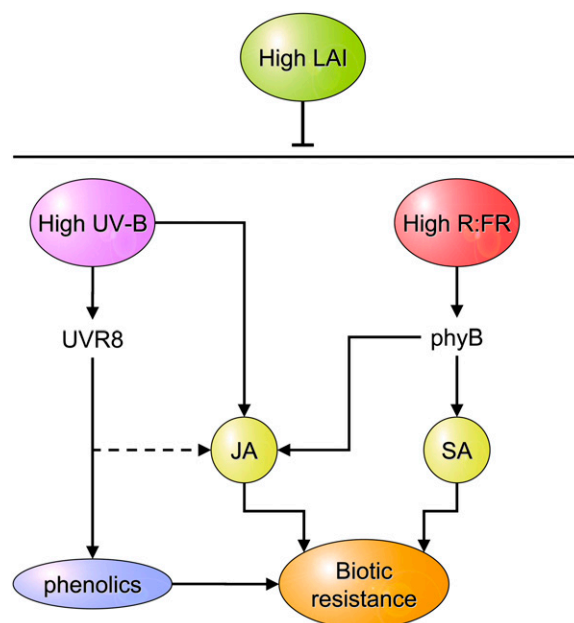


Figure 5. Schematic representation of the positive effects of light signals of open space (high R:FR and UV-B) on plant health. UV-B affects plant resistance to insects and necrotrophic pathogens via mechanisms that involve interactions with JA signaling and JA-independent increases in the levels of certain phenolic compounds, the latter being mediated by UVR8. High R:FR values act through *phyB* increasing plant responses to JA and SA. Dashed lines indicate interactions that have not been explicitly demonstrated. For further explanation, see text.

is also evidence that cryptochrome1 (*cry1*) is a positive modulator of *Arabidopsis* defense against *P. syringae* pv *tomato* DC3000 when plants are exposed to continuous light after pathogen infection (Wu and Yang, 2010). However, other studies, carried out under day/night light cycles, failed to detect any effects of mutations in B photoreceptors on *Arabidopsis* resistance to this bacterial pathogen (Griebel and Zeier, 2008; Jeong et al., 2010). Moreover, *cry1* mutants did not show any obvious susceptibility phenotype in bioassays with the necrotrophic fungus *B. cinerea* (Cerrudo et al., 2012). Therefore, while the participation of cryptochromes in the regulation of SAS responses at high planting density is well documented (Pierik et al., 2004, 2009; Sellaro et al., 2010; Keller et al., 2011; Keuskamp et al., 2011), the effects of variations of B levels caused by the proximity of neighbors on plant immune responses requires further study.

CONCLUSION

In order to achieve elevated yields per unit area, plants must be grown at high density, but increasing crop density and LAI may have negative effects on plant resistance to pests and diseases that are reminiscent of the effects of sunlight deprivation on human health. There is now ample evidence that light, and light signals associated with open space, are positive regulators of plant defense against a broad spectrum of enemies via mechanisms triggered by specific photoreceptors for UV-B and R:FR (Fig. 5). From an evolutionary perspective, this beneficial effect of light might reflect the activity of an optimization strategy that distributes limited resources between growth and defense as a function of the risk of competition that the plant senses using its photoreceptors (Ballaré, 2009). Whether the plant's solution to this dilemma could be manipulated in species of economic interest to reduce pesticide loads without greatly forfeiting crop yields requires further investigation.

The molecular mechanisms that mediate the effects of photoreceptor signals modulating the expression of plant defenses are beginning to be elucidated. Understanding these mechanisms may allow us to manipulate planting density and canopy structure to optimize light penetration for improved crop health. In addition, this understanding will provide key functional information for the design of crop varieties that maintain elevated levels of defense even at high planting density. In this regard, the rapid growth that we have witnessed in the last few years in the field of regulation of plant immunity suggests that, in the not very distant future, we will be able to identify targets for biotechnological manipulation to improve crop health at high LAI. These strategies may help us design agroecosystems that safely deliver healthy products to meet the nutritional demands of humankind in the following decades.

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