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Modulation of plant immunity by light, circadian rhythm, and temperature

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Plants perceive and integrate intrinsic and extrinsic signals to execute appropriate responses for maximal survival and reproductive success. Plant immune responses are tightly controlled to ensure effective defenses against pathogens while minimizing their adverse effects on plant growth and development. Plant defenses induced in response to pathogen infection are modulated by abiotic signals such as light, circadian rhythm, and temperature. The modulation occurs on specific key components of plant immunity, indicating an intricate integration of biotic and abiotic signals. This review will summarize very recent studies revealing the intersection of plant defenses with light, circadian rhythm and temperature. In addition, it will discuss the adaptive value and evolutionary constraints of abiotic regulation of plant immunity.

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Introduction

To survive and reproduce successfully, plants must respond and adapt to their environment. Often plants experience multiple competing developmental and stress signals, and consequently partition and prioritize the use of energy and resources among growth/development, abiotic responses, and biotic defenses. In general, plant immune response against pathogens is a tightly controlled process wherein defense responses are only turned on upon perception of pathogen. This in turn minimizes the adverse effects of defense on growth and development.

Plants employ multi-layered mechanisms to detect and combat pathogens, including preformed physical barriers as well as responses upon recognition of pathogen-derived features (Figure 1). One mechanism recognizes features shared by various pathogens such as PAMP (Pathogen Associated Molecular Pattern) and is named PTI (PAMP Triggered Immunity). A second mechanism recognizes specific pathogen effector(s) and is named ETI (Effector Triggered Immunity) [1,2,3]. These specific pathogen effectors are recognized by the host proteins encoded by *R* (*Resistance*) genes, and a majority of these are nucleotide binding-leucine rich repeat (NB-LRR) class of proteins [4,5,6]. Activation of R proteins often induces local cell death, production of salicylic acid (SA), and leads to induction of systemic acquired resistance (SAR) [[7,8] and a review in this issue]. Yet another mechanism recognizes aberrant RNAs derived from viruses, and this RNA silencing mechanism is employed to defend against viruses by degrading their RNA products [9,10].

Interestingly, plant defense against pathogens is significantly influenced by environmental factors such as temperature, water availability, humidity, light as well as circadian rhythm. This suggests that various environmental signals likely feed into immune responses to balance growth and defense. At present integration and intersection between abiotic and biotic responses remains unclear. This review summarizes recent studies that focus on the molecular mechanisms underlying modulation of plant defense by abiotic factors especially light, circadian rhythm, and temperature. The ecological and evolutionary implications as well as the hormonal mediation of environment signals can be found in earlier reviews [11,12,13].

Light regulation of plant immunity

Involvement of photosynthesis and photoreceptor signaling

Light has a profound effect on plant immunity, and full activation of defense responses to various pathogens is often dependent on light [14,15,16]. A few recent genetic studies confirmed the earlier notion that light dependency of plant immunity is mediated by photosynthesis as well as photoreceptor signaling [14] (Figure 1). For instance, an *Arabidopsis* mutant impaired in nonphotochemical quenching of photosynthesis shows a decreased response to some aspects of PTI but an increased response in other [17•]. Similarly, silencing five individual photosystem II components in *Nicotiana benthamiana* impairs photosynthesis and increases susceptibility to *turnip mosaic virus* [18]. Likewise, resistance to bacterial pathogen *Pseudomonas syringae* (*P.s.*) conferred by the *R* gene *RPS2* (*Resistance to P.s. protein 2*) and *RPM1* (*Resistance to P.s. pv maculicola 1*) is dependent on the red and far-red light receptors PHY (Phytochrome) A and PHYB [19,20]. In rice, resistance to the blast fungus is depend-

ent on PHYA, PHYB, and PHYC [21]. Recently, blue light photoreceptor CRY1 (Cryptochrome 1) was shown to play a critical role in *RPS2*-mediated resistance under continuous light [22•].

The molecular basis of photoreceptor-mediated light signaling was revealed by studies on *Arabidopsis-turnip crinkle virus* (TCV) pathosystem, where resistance to TCV is conferred by the *R* gene *HRT* (*HR to TCV*) [23,24,25••]. Blue light photoreceptors CRY1, CRY2, PHOT (Phototropin) 1, and PHOT2, but not phytochromes, are required for HRT-mediated resistance to TCV. CRY2 and PHOT2 are required for stability of HRT under normal light. HRT is degraded in plants exposed to blue light, which in turn reduces the CRY2 protein level. HRT is degraded in a 26S proteasome dependent manner, and this process might be mediated by an E3 ligase COP1 (Constitutive Photomorphogenesis 1), which interacts with HRT. Thus, photoreceptors might play an important role in R protein stability as well as downstream signaling. Like HRT, the R protein RPS2 also interacts with PHOT2, although a mutation in *PHOT2* does not compromise RPS2-mediated resistance [23,24,25••]. Cyrptochromes and phytochromes are also required for the expression of SA-regulated genes as well as SAR [19,20,22•]. More work is required to unravel light-mediated regulation of R-mediated signaling and their effect on the SA pathway.

Quantity and quality of light

The length of light exposure has a large impact on plant immunity. Daytime inoculation of *P.s. pv. maculicola* (*Psm*) with effector *AvrRpm1* triggers a more robust defense response than night time inoculation, possibly because of the dependence of SA accumulation on the length of light exposure [20]. The effect of length of light exposure on SAR was proposed to explain discrepancy in the role of methyl salicylate (MeSA) in SAR [26•,27]. MeSA is required for SAR development when plants receive little or no but not long exposure to light after being infected by an avirulent pathogen. Thus the length of light exposure can change the relative roles of different defense pathways or molecules in plant immunity. This regulation of immunity by day length or light length could act through different light signaling components. For instance, the blue light receptor *CRY1* is required for defense responses against bacterial pathogens under continuous light but not under short day conditions [20,22•].

Plant immunity is also regulated by light quality [15]. This modulation mostly functions through sensing the Red/Far Red (R/FR) ratio to regulate the jasmonate acid (JA) pathway [28]. The *phyB* mutant, which is unable to perceive red light, is more susceptible to the fungal pathogen *Fusarium oxysporum* than wild type [28]. Similarly, a low R/FR ratio reduces resistance to the necrotrophic pathogen *Botrytis cinerea* in *Arabidopsis*. This effect of the R/FR ratio on this resistance is independent of SA but is dependent on the JA signaling components COI1 (Coronatine-insensitive 1) and JAZ10 (Jasmonate ZIM domain) [29•], demonstrating a critical role of JA signaling in conveying the light quality signal.

Outside of the visible spectrum, UV-B radiation at low doses increases plant resistance to *B. cinerea*. Genetic studies revealed that this effect is conferred by UVR8, a UV-B photoreceptor [30•]. Further, the UV-B effect was independent of the common defense molecules such as JA, glucosinolates, or camalexin, but dependent on ferulic acid 5-hydroxylase for sinapate biosynthesis [30•]. Thus low doses of UV-B may enhance the synthesis of syringyl-type lignin, which is involved in fortification of cell wall that acts as a physical barrier.

Therefore, light executes its modulation on plant immunity through its many attributes. Various aspects of light signals are transduced in plants by distinct light signaling pathways to modulate multiple components and processes in plant immunity (Figure 1).

Circadian regulation of plant immunity

A circadian clock regulation of plant immunity was previously suggested based on the observation that some defense related genes and stomata closure are modulated in a circadian manner [14]. Recent results have established an important role for circadian rhythm in defense against microbial pathogens [31••] (Figure 1). In this study, promoters of genes required for PTI and ETI were found to be enriched with evening elements that are regulated by the clock. The loss of circadian function by the *cca1* (*circadian clock associated 1*) mutation compromised resistance to the downy mildew while *CCA1* overexpression enhanced resistance to this pathogen. Furthermore, wild-type plants but not the *cca1* mutants are more susceptible to downy mildew when infected at dusk than at dawn, indicating a role for the clock gene *CCA1* in enhancing resistance at dawn [31••]. Circadian regulation has also been demonstrated in the interaction of *Arabidopsis* with an avirulent *P. syringae* strain [32•]. In free-running light conditions, wild-type plants display greatest susceptibility at the subjective midnight and greatest resistance at the subjective morning as perceived by the clock. This temporal variation in resistance is lost in the clock mutant *elf3* and the transgenic line overexpressing *CCA1*. In addition, most of the central genes mediating PTI have peak expression in the subjective morning, which is likely responsible for the circadian regulation of PTI.

Resistance against herbivores is also regulated in a circadian manner [33••]. *Arabidopsis* plants show higher resistance to cabbage loopers when both are clock-entrained in phase than out of phase. Both circadian clock function and JA are required for this defense enhancement because