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Invited Review

Green Light to Plant Responses to Pathogens: The Role of Chloroplast Light-Dependent Signaling in Biotic Stress†

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ABSTRACT

Light has a key impact on the outcome of biotic stress responses in plants by providing most of the energy and many signals for the deployment of defensive barriers. Within this context, chloroplasts are not only the major source of energy in the light; they also host biosynthetic pathways for the production of stress hormones and secondary metabolites, as well as reactive oxygen species and other signals which modulate nuclear gene expression and plant resistance to pathogens. Environmental, and in particular, light-dependent regulation of immune responses may allow plants to anticipate and react more effectively to pathogen threats. As more information is gathered, increasingly complex models are developed to explain how light and reactive oxygen species signaling could interact with endogenous defense pathways to elicit efficient protective responses against invading microorganisms. The emerging picture places chloroplasts in a key position of an intricate regulatory network which involves several other cellular compartments. This article reviews current knowledge on the extent and the main features of chloroplast contribution to plant defensive strategies against biotic stress.

INTRODUCTION

The evolutionary history of plants is shaped by interactions with a changing environment and with a plethora of viral, bacterial and fungal pathogens, which feed on photosynthetic end products. Plants represent the motionless party in these biotic interactions and therefore, they have evolved sophisticate biochemical and physiological mechanisms to sense, signal and respond to the challenges of attacking microorganisms, deploying defensive barriers that pathogens need to overcome in order to colonize the host and spread disease (1–3). While plant susceptibility or resistance to pathogen infection has genetic determinants, the degree of tolerance is also influenced by the plant age, the nutritional status and the environmental conditions. Among the latter, light plays a major role in the final outcome of the interaction: survival or disease.

Light is the basic energy source for production of biomass through photosynthetic electron transport, but it also provides signals to control plant development and to deploy successful defensive responses against environmental stresses, including pathogens. Plants can sense many different properties of light, such as intensity, amount, quality and duration of the photoperiod, each of them with its own regulatory networks, and are also subject to circadian regulation. Families of photoreceptors, spanning from the near-ultraviolet to the far-red regions of the spectrum, perceive and integrate different qualities of light, and utilize this information to execute appropriate responses for maximal survival and reproductive success (4). The intensity and total amount of light also impact directly on chloroplasts and the photosynthetic apparatus. At least three different types of light-dependent perturbations have been identified that initiate plastid-associated responses: (1) changes in the redox status of the photosynthetic electron transport chain (PETC), (2) increased production of singlet oxygen ($^1O_2$) and (3) enhanced generation of partially reduced forms of oxygen, such as superoxide ($O_2^{-\cdot}$) and hydrogen peroxide (H_2O_2). These three oxygen derivatives, together with the hydroxyl radical ($OH^{\cdot}$), are collectively known as reactive oxygen species (ROS) and can react with many different types of biomolecules. They can also act as environmental cues to trigger defensive responses (5).

In the case of biotic interactions, and by comparison with mammalian systems, much attention was initially devoted to ROS generation in the apoplastic space by nicotinamide adenine dinucleotide, reduced form (NADPH) oxidases bound to the plasma membrane. Subsequent observations, however, indicated that light-dependent ROS propagation from the chloroplasts also plays a key role in some critical aspects of the defensive strategy deployed by the plant to face an invading microorganism. This review provides a brief overview of light and ROS signaling during plant–pathogen interactions and focuses on the contribution of chloroplasts to the common effort displayed by the host to prevent colonization and disease.

OVERVIEW OF IMMUNE DEFENSE IN PLANTS

Plant pathogens are usually classified as biotrophs, hemibiotrophs or necrotrophs, depending on whether they require living host

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cells to propagate infection (biotrophs), at least during the early stages of the interaction (hemibiotrophs), or induce necrosis to feed on the collapsing host tissue (necrotrophs) (6,7). While similar mechanisms are presumably utilized in the recognition of all classes of pathogens, the responses deployed by the infected plant will be forcibly different depending on the lifestyle of the microorganism. Indeed, plants elicit multilayered responses to detect and combat pathogens, including preformed defenses as well as responses induced only after recognition of the microbe (Fig. 1).

For a pathogen to gain access to plant cells, it must first overcome a series of passive barriers that the host displays as preformed obstacles, such as wax layers and rigid cell walls. When the invading microbe manages to advance through these constitutive defenses, it becomes a potential target for recognition by the plant (7). Molecular signatures conserved within classes of microorganisms, such as flagellar proteins or exopolysaccharides (called pathogen-associated molecular patterns or PAMPs), are sensed through specific membrane-bound receptors belonging to the pattern recognition receptor (PRR) family (Fig. 1). PRRs display intracellular protein kinase activity to signal the presence of the pathogen. These early signaling events initiate mechanisms of PAMP-triggered immunity (PTI), involving a massive reprogramming of gene expression to elicit specific protective reactions in the infected plant (1,2,7,8). They include redirection of metabolism from growth to defense, increases in ROS production, activation of hormonal signaling, sealing of infected cells by callose deposition into the cell wall, and biosynthesis of antimicrobial secondary metabolites (8). By combining these various means of defense, plants attempt to prevent the microbe from colonizing the tissue, leading, if successful, to a type of resistance named non-host (Fig. 1). In a non-host interaction, all members of a given plant species are in principle resistant to a whole class of microorganisms (3). As multiple PAMPs and receptors are usually involved in PTI, non-host resistance is multigenic.

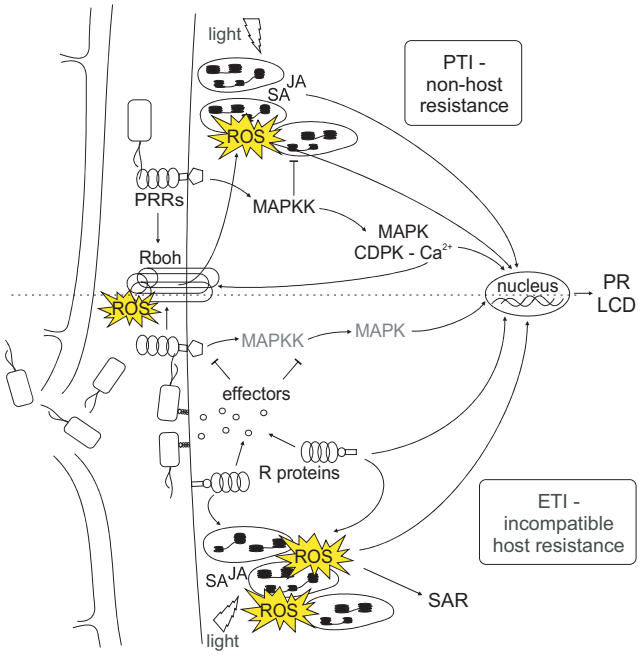

Figure 1. Simplified model describing different types of interaction between plants and invading microorganisms. In the upper part, non-host pathogens which are able to penetrate the constitutive barriers of the plant cell (*e.g.* cell walls) are recognized through their pathogen-associated molecular patterns (in the example, flagella) by pattern recognition receptors (PRR) bound to the plasma membrane, which will ultimately result in the establishment of PAMP-triggered immunity (PTI). This process is signaled via mitogen-activated protein (MAP) kinase (MAPK-MAPKK) and calcium-dependent protein kinase (CDPK) activities, and involves reactive oxygen species (ROS) production by NADPH oxidase (Rboh) and chloroplast metabolism, as well as increased synthesis of SA and JA. In the lower part, evolution of effector genes by individual pathogen strains or races allows them to dodge or evade PTI and propagate disease. Individual plant lines, in turn, evolved genes encoding R proteins to neutralize these effectors, resulting in development of effector-triggered immunity (ETI). In many instances, PTI and ETI lead to reprogramming of nuclear gene expression which results in localized cell death (LCD) at the site of infection and induction of pathogenesis-related (PR) genes. Other details are given in the text.

In the course of evolution, certain strains of a given pathogen were able to evolve or acquire virulence elements (*avr* genes, whose products are also termed effectors) that could be delivered into the plant cell and suppress or circumvent PTI reactions, usually by targeting one or more of their components (7). The affected plant then became a host and could be infected by the virulent isolate, while not by other strains or races of the same microbial species (Fig. 1). In response to this challenge, specific lines of the host evolved novel products (encoded by the *R* genes) which could interact directly or indirectly with the *avr* products and still activate an immune response, in this case termed effector-triggered immunity (ETI). The plant line then became resistant to a given pathogen isolate in the context of an incompatible host interaction called avirulent (Fig. 1; see also 7,9). This type of resistance depends on the presence of individual genes (*R* and *avr*) in both the plant and the microbe, and is therefore inherited in a mendelian way. For this reason, it is also called gene-for-gene resistance (10).

Genome-wide analyses have shown that many common responses are activated during both PTI and ETI (11, 12). These results suggest that while acquisition of a new class of recognition molecules (the R proteins) was necessary for evolution of ETI, plants used most of the pre-existing PTI machinery for defense (11). Still, immune responses triggered during ETI are reported to be more prolonged and robust than those observed in PTI (7,11–13), indicating that at least part of the differences between the two types of responses can be quantitative. It should be born in mind, within this context, that PAMPs are also present in non-pathogenic microorganisms, and given this low microbial recognition specificity of PTI, it may be beneficial for plants not to induce, at an early stage of the interaction, a strong immune response that could negatively affect their fitness. This notion implies that plants must have mechanisms to evaluate the effect of the initial PTI response and amplify the immune signals only when a stronger response is required (11). While this argument provides a rationale to explain the comparative features of the two immune systems, the genes and mechanisms that make ETI a stronger and faster version of PTI are still unknown.

In general terms, resistance against biotrophic pathogens mainly involves pathways signaled by salicylic acid (SA), whereas mechanisms modulated by jasmonic acid (JA) and ethylene act against necrotrophic microorganisms. The SA and JA/ET regulations have been usually regarded as mutually antagonistic, but the existence of synergistic interactions has also been reported (13–15). In many instances, resistance mediated by both

PTI and ETI culminates in localized cell death (LCD) in the vicinity of the pathogen entry site. In the case of ETI, this LCD is part of a hypersensitive reaction (HR) which also involves induction of various pathogenesis-related (PR) genes, and is proposed to contribute to the containment of biotrophic microorganisms by opposing a barrier of dead cells to deter their spread into the adjacent tissue. The role played by LCD during infection by necrotrophic pathogens is less understood, since suicidal tissue death would simply make their life easier.

As indicated, LCD can be observed during non-host biotic interactions (see for instance, ref. 16), and also in plant responses to a number of abiotic stresses such as exposure to ozone (17), and excess of excitation energy (EEE), a condition in which the amount of light absorbed by the photosystems (PS) exceeds that required for photosynthetic metabolism (18).

The link between ROS and the HR was established more than 30 years ago, when Doke (19) reported superoxide production prior to the HR elicited by *Phytophthora infestans* and tobacco mosaic virus on potato and tobacco, respectively. A biphasic oxidative burst commonly precedes cell death, and the signaling role played by these oxidants in the orchestration of the HR has long been recognized (20,21). ROS are also involved in propagation of the defensive response to tissues located far from the affected site, leading to systemic acquired resistance (SAR) and acclimation (SAA) during biotic and abiotic interactions, respectively (22–24). Despite this wealth of information, many aspects of ROS function remain obscure. For instance, it is not clear if they participate in triggering LCD, in the induction of PR genes, or in both pathways. Also, the relative contribution of ROS produced in different compartments has yet to be established.

In addition to ROS, full manifestation of the defense response in plants has been shown to be affected by light, as attenuated responses to a number of bacterial, fungal and viral pathogens have been frequently observed in the dark (25–27). In the following sections, we will review the available information concerning the role of illumination during plant responses to biotic stress, and its relationship with ROS generation and signaling.

MORE LIGHT TO DETER PATHOGEN INVASION: PHOTORECEPTORS

As early as 1970, Lozano and Sequeira (28) recognized that plant responses to pathogen invasion were markedly different under light or dark conditions. Light has been shown to play important roles in SA-mediated pathways, and to be required for the HR (29). Plants infected in the dark show reduced lesion formation in response to non-host and avirulent pathogens (26,27,30–33). SAR has also been shown to be light-dependent (34). Lack of a carbohydrate source is not the limiting factor that restricts these responses in the dark or in dim light, suggesting that they do not depend on photosynthetic products but rather on light sensing and signaling (29,34).

Several classes of photoreceptors have been identified in *Arabidopsis* and other species, although the search for light sensors is still ongoing in higher plants. Photoreceptors influence many developmental and environmental responses during plant life (4,35). Phytochromes are the best studied among them: dimeric chromoproteins containing a covalently bound linear tetrapyrrole (bilin) at the N-terminal region of each subunit. They constitute a family of five genes in *Arabidopsis* (*phyA-phyE*) and sense mostly red and far-red light (36). Upon illumination with the proper wavelength, they migrate to the nucleus and modulate the expression of many light-responsive genes (Fig. 2). A link between phytochrome-mediated signaling and the defense response against biotic stress has been proposed, as a low red/far-red ratio reduces resistance to the necrotrophic fungus *Botrytis cinerea* in *Arabidopsis* (37), and a *phyB* mutant line was more susceptible than its wild-type (WT) parental to *Fusarium oxysporum* infection (38). Moreover, *phyA phyB* double mutants displayed reduced HR and increased susceptibility to *Pseudomonas syringae* pathovar (pv) *tomato*, an avirulent strain (29). However, subsequent studies failed to find an analogous role for phytochromes in ETI against turnip crinkle virus or avirulent *P. syringae* pv *maculicola* (27,33), although *phyA phyB* double mutants were compromised in the establishment of SAR (33).

Two other known plant photoreceptors, cryptochromes and phototropins, scan the blue and the UV-A regions of the spectrum. Both contain flavin, and cryptochromes harbor in addition a light-harvesting pterin and a methenylotetrahydrofolate molecule (34). They are reported to undergo light-dependent mobilization into the cytosol, either from the plasma membrane or the nucleus in the case of phototropins and cryptochromes, respectively (Fig. 2). Initial observations suggested that they did not play any significant role in plant immunity (33). Further research, however, showed that the contribution of the *Arabidopsis* cryptochrome CRY1 was dependent on the environmental conditions. Wu and Yang (39) used *cry1* mutants and CRY1-overexpressing lines to show that the blue light receptor did contribute to resistance to an avirulent *P. syringae* strain when plants were

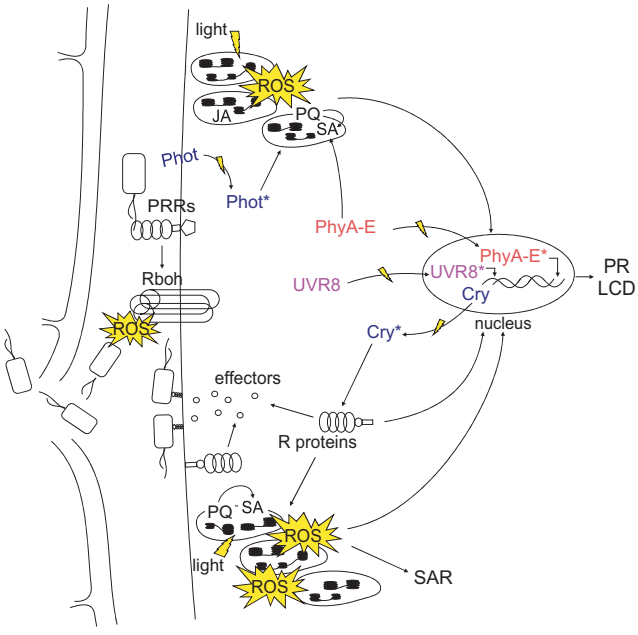

Figure 2. Interplay between light, reactive oxygen species (ROS) and biotic resistance. Light-sensing components are described schematically. Upon exposure to light of the corresponding wavelength (see text), phytochromes (PhyA-E) and UVR8 move from the cytosol to the nucleus, whereas cryptochromes (Cry) follow the opposite pathway and phototropins (Phot) dissociate from the plasma membrane into the cytosol. The activated forms of these photoreceptors, indicated by asterisks, influence nuclear gene expression and other cellular processes. Light also affects chloroplast metabolism and signaling defensive responses via ROS build-up and the redox status of plastoquinone (PQ). For the sake of clarity, the interplay of light and ROS with hormone signaling has been omitted.

exposed to constant light, rather than the short day conditions employed in previous studies (33).

Outside the visible spectrum, exposure to UV-B radiation increased *Arabidopsis* resistance to *B. cinerea*, in a process that was independent of common defense molecules such as JA or phytoalexins (40). Genetic studies revealed that this effect was mediated by UVR8, a UV-B photoreceptor (40).

The length of light exposure also has a large impact on plant immunity (41). Daytime inoculation of avirulent *P. syringae* pv *maculicola* triggers a more robust defense response than nocturnal inoculation, possibly because of the dependence of SA accumulation on the length of light exposure (33). This regulation of immunity by day length or light length could act through different light signaling components, as already described for CRY1. Light availability is particularly important during the first hours after inoculation, as the absence of light at the early phase of a plant–pathogen interaction negatively affects development of resistance at later stages (33).

It is worth noting, however, that not all inducible plant defenses are dependent on illumination. In *Arabidopsis* leaves inoculated with an avirulent *P. syringae* strain, darkness did not affect biosynthesis of the host phytoalexin, camalexin, JA accumulation, or expression of GST1, a ROS-responsive glutathione *S*-transferase (26).

QUANTITY OF LIGHT: THE CHLOROPLAST CONNECTION

Genoud *et al.* (29) were the first to report that functional chloroplasts are required for the HR, suggesting the existence of a signaling pathway, different from that of photoreceptors, which links plant immunity with light perception through plastid metabolism. Indeed, chloroplasts have the potential to act as delicate environmental sensors, as they harbor numerous pathways that are readily unbalanced by environmental fluctuations, and the photosynthetic apparatus constitutes a light-sensing system in its own right. In addition, plastids also contribute to plant immunity by hosting diverse metabolic pathways whose products participate in stress resistance and signaling. They include production of nitric oxide and biosynthesis of SA and JA (42).

Allocation of resources for defense and biosynthesis of protective compounds causes a demand for energy in the infected tissue. However, several studies have reported that photosynthesis is downregulated in response to various types of pathogens, forcing plants to shift toward non-assimilatory metabolism (43,44). Collapse of photosynthetic activity leads to a metabolic transition from source to sink in infected tissues. The resulting demand for carbohydrates and energy becomes compensated through increased activities of cell wall invertases, hexose transporters, the oxidative pentose phosphate pathway and respiration (45). Such reprogramming of primary carbon metabolism is expected to favor production of secondary compounds with antimicrobial activity (43).

Malfunction of the photosynthetic apparatus results in perturbations of the chloroplast redox status and ROS build-up. Both mechanisms can be used as signals to instruct defensive responses. Once again, the situation resembles that of plants exposed to EEE. Indeed, plant responses to EEE have a number of striking parallels with the immune response, including ROS production, SA-mediated signaling, the formation of lesions via LCD, and the expression of a number of common genes, such as the PR markers PR2 and GST6 (18,46,47). Moreover, plants acclimated to EEE displayed increased resistance against a virulent *P. syringae* strain (18,46). This very interesting aspect of the relationship between light stress and immunity is beyond the scope of this review. The reader is referred to the excellent article by Karpiński *et al.* (24) for further reading.

Treatments with light enriched at different wavelengths and with inhibitors that block photosynthetic electron transport at various sites of the chain have shown that the redox poise of the plastoquinone pool appears to play a key role in plant responses to both excess light and pathogen attack, especially in LCD regulation and in initiation of systemic responses (24,34,48). Evidence also indicates that ROS generated in chloroplasts can provide information to initiate LCD during non-host and avirulent interactions. Photosynthetic electron transfer reactions represent the major source of ROS in chloroplasts (and leaf cells) due to the generation of strongly reactive species during primary photochemistry. In the reaction center of PSII, $^1O_2$ is produced via reaction between excited triplet-state chlorophyll from the P680 reaction centers and molecular oxygen (49,50). Oxygen may also drain electrons from several sites of the PETC, notably the highly reducing components in and after PSI, leading to the formation of $O_2^{-\cdot}$ and H_2O_2 in the stroma (51). These two ROS can inactivate (to various extents) many different proteins and lipids (5,52), whereas $^1O_2$ is reported to account for more than 80% of non-enzymatic lipid peroxidation in chloroplasts (53). While the deleterious effects of ROS in chloroplast metabolism have been extensively characterized, their transient increases during episodes of biotic or abiotic stress might be beneficial in terms of signaling effects against the very same conditions that induced their accumulation (23,54). So far, H_2O_2 is the best characterized signal involved in this chloroplast–nucleus communication, or "retrograde signaling", although the mechanistic aspects of its action remain largely unknown.

Chloroplasts contain a suite of overlapping antioxidant systems. While chemical antioxidants such as glutathione, ascorbate and tocopherols can in principle inactivate any ROS, enzymatic scavenging is only efficient with the less reactive species, namely $O_2^{-\cdot}$ and H_2O_2. Several superoxide dismutases, peroxidases (with different substrate specificities), catalases and enzymes involved in antioxidant synthesis and regeneration are called into action to counterbalance ROS production and maintain their concentrations below dangerous levels (55). Expression of these proteins also responds to environmental stimuli, resulting in a dynamic interaction between ROS production and scavenging (56). It has been argued that transient inactivation of the ROS scavenging processes may be as important as increased ROS production in triggering specific ROS-dependent signaling responses within the cell (57).

The extent of the impact of chloroplast ROS on immune reactions has been explored using transgenic tobacco plants expressing a plastid-targeted cyanobacterial flavodoxin. Flavodoxin acts as a general antioxidant specific for chloroplasts, preventing over-reduction of the PETC and electron delivery to oxygen, which results in low levels of ROS in plastids (58). These transgenic plants displayed attenuated LCD upon infection by a non-host pathogen, whereas neither the synthesis of SA or JA nor the expression of PR genes were affected by the presence of the flavoprotein (16). Conversely, silencing of chloroplast enzymes that scavenge plastidic H_2O_2 led to spreading of LCD in response to coronatine, a phytotoxin produced by virulent strains

of *Pseudomonas* (59). This effect of chloroplast-generated ROS on pathogen-triggered LCD appears to be mediated by a mitogen-activated protein kinase cascade (32).

Studies of mutants have confirmed the contribution of photosynthetic electron transport and light-induced ROS production to the onset of LCD in response to bacterial pathogens (60,61). Much of this understanding has been obtained by utilizing lesion-mimic mutants that show enhanced HR-like cell death under stress (61). One of the best known examples is the lesion simulating disease 1 (*lsd1*) mutant, which fails to limit the spread of the HR, and undergoes runaway cell death when infected by avirulent pathogens or upon exposure to EEE (24,30,34,62). This effect has been linked to failure of *lsd1* plants to upregulate genes encoding Cu/Zn superoxide dismutase and catalase 1, which act as antioxidant enzymes in chloroplasts and peroxisomes, respectively (30).

In addition to the PETC, $^1O_2$ can also be generated in chloroplasts during chlorophyll metabolism. The chlorophyll biosynthesis mutant *fluorescent* (*flu*) has provided an elegant model to study $^1O_2$ signaling (63). This mutant accumulates the chlorophyll precursor protochlorophyllide in the dark, and shows enhanced $^1O_2$ synthesis upon re-illumination. As light-dependent $^1O_2$ production is not sufficient *per se* to induce the LCD response in *flu* seedlings, Lee *et al.* (63) selected second site suppressors. This led to the identification of EXECUTER 1 and 2, two chloroplast-targeted and thylakoid-bound proteins, which control $^1O_2$ synthesis responses (64), together with cryptochrome CRY1 (65). A microarray analysis of dark-adapted WT and *flu Arabidopsis* plants exposed to light showed that the FLU-mediated $^1O_2$ signaling pathway shares many common features with the stress responses induced by pathogen attack, wounding and abiotic stresses. Moreover, $^1O_2$-mediated LCD occurs only in cells containing fully developed chloroplasts (66), in line with the observations of Genoud *et al.* (29). Given the many contributions of chloroplasts to the defense against biotic stresses, it is not surprising that many bacterial effectors directly target chloroplastic functions (67).

INTERPLAY BETWEEN PLASTIDIC AND EXTRACHLOROPLASTIC ROS SOURCES DURING PLANT RESPONSES TO BIOTIC STRESS

Chloroplasts are not the only source of ROS in the plant cell, and ROS signaling effects may arise as a result of crosstalk between different cellular compartments (23). In the plasma membrane, the flavoenzyme NADPH oxidase, encoded by the *rboh* family, translocates electrons from cytosolic NADPH to extracellular oxygen, leading to the generation of $O_2^{-\cdot}$ and H_2O_2 in the apoplast (68). Hydrogen peroxide can also be produced in the same compartment by the activity of an extracellular polyamine oxidase isoform (69). Knocked-down plants with decreased levels of this enzyme failed to develop cell death upon induction with the elicitor cryptogein (69).

Activation of NADPH oxidases mediates the progression of ROS signals from cell to cell (70,71). At first sight, it might seem odd that pathogen attack induces ROS production by a committed enzyme family of the host at a time in which ROS are generated by adventitious reactions (photosynthesis, respiration) in various other cell compartments, and several members of the antioxidant response are also activated to counteract ROS build-up. Analysis of the evolution of ROS-related genes along the plant lineage sheds light on the life history of ROS production, scavenging and signaling in *Viridiplantae*. Using genomic data from dicots, monocots, vascular non-seed plants, mosses and green algae, Mittler *et al.* (55) reconstructed the ancestral set of ROS-related genes ("the ROS network") at different time points during evolution, and traced back the origin of newly acquired traits. This analysis revealed that, except for catalases, all genomes of the plant lineage encode members of each ROS scavenging family (superoxide dismutase, ascorbate peroxidase, glutathione reductase, etc.). In contrast, a complete absence of the ROS-producing NADPH oxidase gene family was observed in the algal genomes. Only from mosses on, plants appeared to have acquired genes belonging to this family. This evolutionary novelty might have been associated with the need for a more complex signaling network to coordinate multicellular growth, morphological complexity and stress responses. The analysis suggests that ROS scavenging mechanisms preceded ROS-producing mechanisms, and that plants first learned to control their intracellular ROS levels and only then started to use ROS for signaling purposes (55).

The apoplastic ROS burst mediated by NADPH oxidase can also trigger ROS production in chloroplasts (17,72). This is structurally feasible, because under high light conditions chloroplasts adopt a position adjacent to the plasma membrane, facilitating communication between compartments (23).

In addition from chloroplasts and the apoplastic space, ROS can be produced in mitochondria, peroxisomes and even the nucleus (73). While mitochondria dominate ROS generation in animal cells, the role of these organelles in plant ROS production is subtler. The mitochondrial enzyme proline dehydrogenase (ProDH) has been proposed to contribute to HR development and disease resistance, and to potentiate ROS production in mitochondria by delivering reducing equivalents to the respiratory chain. Indeed, *proDH*-silenced plants accumulated less ROS and developed reduced cell death in response to an avirulent pathogen (74). The ROS contribution of other cellular sources appears to be marginal and is not addressed in this review.

As incomplete as this information might seem, even less is known on the systems and mechanisms put into action to sense the different ROS produced in the various compartments. Noteworthy, comparison of the transcriptional responses to H_2O_2 generated in chloroplasts, in peroxisomes or in the apoplast revealed little similarity in the patterns of gene expression (75–77). Within the chloroplast itself, H_2O_2 and $^1O_2$ also elicit specific and to some extent antagonistic effects on gene expression (75,78). The overall results indicate that the specific combinations of ROS, as well as their temporal and spatial accumulation, determine ROS specificity and response (77).

CONCLUDING REMARKS

Plants live in an environment with changing abiotic and biotic conditions, and although it is well established that plant defense to pathogen invasion is under genetic control, the success of these protective measures is also influenced by environmental conditions. Among them, illumination plays a central role, and plant biotic interactions are under the multifaceted influence of light, which exerts its effects through light-dependent photosynthetic processes, as well as mechanisms that may respond to light wavelength and daylength. Different light qualities have been

shown to evoke photoreceptor-mediated signaling cascades that ultimately modify nuclear gene expression. The redox status of the PETC, together with ROS homeostasis, also contributes significantly to these processes.

Much of the research on plant responses to biotic challenges has focused on the role played by cellular photoreceptors and ROS produced in the apoplast, but increasing evidence indicates that the chloroplast, as the engine of plant development and growth, also plays a crucial role in both light and ROS signaling. These organelles can sense light intensity via changes in the redox poise of the PETC (notably through plastoquinone), and signal back to the nucleus to elicit responsive changes in gene expression. They house several important steps in the synthesis of SA, JA, abscisic acid and antimicrobials, and are major contributors to the cellular status of sugars, which can regulate gene expression by themselves. Chloroplasts are also the main source of $^1O_2$, $O_2^{-\cdot}$ and H_2O_2, and interact with other ROS-producing organelles such as the apoplast, peroxisomes and mitochondria, to establish a ROS signaling network that triggers an important subset of defense responses. Understanding the complex molecular crosstalk in light and ROS signaling during biotic interactions will be highly relevant in efforts to enhance the stress tolerance of plants for sustainable productivity in the future.

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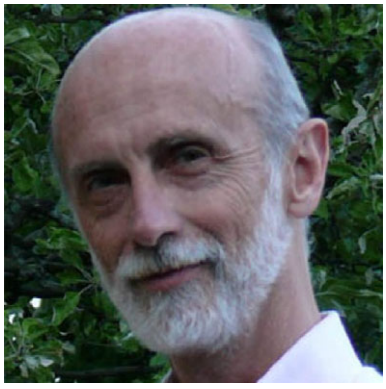

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