Invited Review

Bacterial Photosensory Proteins and Their Role in Plant–pathogen Interactions[†]

Ivana Kraiselburd[‡], Laura Moyano[‡], Analía Carrau, Josefina Tano and Elena G. Orellano*

Instituto de Biología Molecular y Celular de Rosario, Consejo Nacional de Investigaciones Científicas y Técnicas, Facultad de Ciencias Bioquímicas y Farmacéuticas, Universidad Nacional de Rosario, Rosario, Argentina

ABSTRACT

Light is an important environmental signal for almost all living organisms. The light perception is achieved by photoreceptor proteins. As can be observed from the great number of bacterial genomes sequenced, plant pathogenic bacteria encode for a large number of photoreceptor proteins. The physiological implications of these photoreceptors are still poorly characterized. However, recent studies revealed the participation of these photosensory proteins in the pathogenic process. Here, we summarize what is known about these proteins and their role during the virulence process, concluding that the light environment modulates the plant–pathogen interaction.

INTRODUCTION

Light is an important source of energy and information, and the ability to respond to this stimulus is a widely distributed feature in all domains of life. Light is essential for plant metabolism and it modulates almost every aspect of plant physiology, including pathogen attack. On the other hand, the effect of light in the physiology of quimioheterotrophic bacteria is a novel paradigm that has attracted worldwide interest for its potential implications. In living organisms, light sensing is accomplished by photoreceptor proteins carrying structures suitable for light absorption. Biological photoreceptors can be classified into six distinct families based on the structure of their chromophores, that is the molecules responsible for light absorption. Rhodopsin, phytochromes and xanthopsins present a photochemistry based on the *E/Z* isomerization of their associated chromophores, which are retinal, phytochromobilin and p-coumaric acid, respectively. Cryptochromes and proteins with LOV (**L**ight, **O**xygen or **V**oltage) or BLUF (**B**lue **L**ight sensing **U**sing **F**lavin) domains employ a flavin-based photochemistry. Most biological photoreceptors have light perception domains coupled to a variety of signaling domains which are responsible for the transduction of the light signal (1).

LOV domain proteins are photoreceptors sensitive to the blue region of the electromagnetic spectrum. These domains present flavin mononucleotide (FMN) as chromophore, which is noncovalently attached to the protein moiety, but becomes covalently bound upon the absorption of blue light. The covalent bond is formed by means of a conserved cysteine residue, after the light-induced conversion of the chromophore into a triplet state. The covalent form represents the active (signaling) state of the photoreceptor. This form thermally switches back to the dark state of the protein (2).

BLUF proteins are flavin-binding, blue light-sensing proteins found in many bacteria and some algae. This family of photoreceptors uses FAD (Flavin Adenine Dinucleotide) as chromophore. In these proteins, a transient and reversible red-shifted state (BLUF$_{RED}$) is generated upon blue light irradiation, dictated by a hydrogen-bonding switch reaction involving N(5), O(4) and two conserved tyrosine and glutamine residues. BLUF$_{RED}$ formation seems to involve a light-driven electron and photon transfer from a conserved tyrosine residue to FAD, followed by hydrogen-bonding rearrangement and radical pair recombination (3,4).

Red-light-absorbing phytochromes are well known and characterized photoreceptors. Phytochromes were first discovered in higher plants, but they have also been found in cyanobacteria and in nonphotosynthetic organisms. In bacteria, these photoreceptors are commonly called bacteriophytochromes (BphPs). The typical architecture of BphPs consists of an N-terminal photosensory region with three conserved domains: PAS, GAF and PHY, followed by a C-terminal regulatory histidine kinase. BphPs possesses a linear tetrapyrrole bilin (biliverdin IXα) as a chromophore, which is normally covalently attached via a thioether linkage of a cysteine residue. These photoreceptors can be photoconverted between the red-absorbing state (Pr) and the far-red-absorbing (Pfr) one. The Pr form shows an absorption maximum at 700 nm, while the Pfr form absorbs at 750 nm. Light absorption of the Pr form causes a rapid photo-isomerization of the double bond between rings C and D of the chromophore. This photoreaction is followed by conformational changes that generate the Pfr form, where the signal transduction cascade starts (5–7).

The first associations of light perception with bacterial processes correspond to organisms that are capable of coupling light absorption to the generation of energy such as cyanobacteria, performing oxygenic photosynthesis, and green and purple bacteria, developing anoxygenic photosynthesis. However, the discovery of genes encoding photoreceptors in the genomes of nonphotosynthetic and nonphototactic bacteria aroused great interest in studying the potential light regulation of bacterial physiology. In the last decade, numerous studies revealed the

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physiological role of bacterial photoreceptors, especially those sensitive to the blue region of the electromagnetic spectrum both in pathogenic and nonpathogenic bacteria (2). The diverse roles revealed by the photoreceptors present in nonphototrophic bacteria suggest that in these microorganisms, light provides information about their location, enabling them to make lifestyle decisions such as changing from motile to sessile states, starting a response to stress or inducing virulence according to their environmental situation (8). Below, we summarize the characteristics of the most studied photoreceptors present in plant pathogens (Table 1). We focalize our attention in bacterial pathogens, describing the photosensory proteins present in these organisms and their roles in bacterial physiology and pathogenicity.

PARTICIPATION OF LIGHT AND BACTERIAL PHOTORECEPTORS IN PLANT–PATHOGEN INTERACTION

The outcome of plant–pathogen interactions is significantly influenced by environmental factors such as temperature, humidity and light. In addition, plant vulnerability against pathogen attacks as well as the pathogen virulence varies with the stage of development of both organisms and the time of day in which the interaction occurs. In this regard, there is increasing evidence supporting the importance of light in plant–pathogen interactions due to the effects of this environmental stimulus both on plant defense response and on pathogen virulence (9). Regarding light regulation of plant responses against pathogen attack, numerous reports associate light perception with the development of efficient defense responses against viral, bacterial and fungal pathogens, various components of these responses being inhibited in the dark (10–12). While some aspects of plant defenses are independent of light, this stimulus has an important role in responses mediated by salicylic acid, a hormone deeply involved in plant immunity (13). While the light-induced accumulation of this hormone depends on the specific plant and pathogen, the light requirement for triggering salicylic acid-dependent responses is a general aspect of plant–pathogen interactions (10,11,14,15). Although the molecular mechanisms involved in the light dependency of plant immune responses is not fully understood, it is known to be dependent on components of plant photosynthesis as well as on photoreceptors of red/far-red light (phytochromes) and blue light (cryptochromes and phototropins) (9). Apart from being influenced by the quality of light, plant defense responses are affected by the quantity of light. In this regard, Griebel and Zeier demonstrated that within a fixed photocycle, resistance responses of *Arabidopsis thaliana* plants against a virulent strain of *Pseudomonas syringae* depend on the time of the day when the infection occurs (plants were grown in a controlled chamber; photon flux density 70 μE m^{-2} s^{-1}; day period temperature: 21 °C, night period temperature: 18 °C) (11). Morning inoculations resulted in higher accumulation of salicylic acid, a faster expression of pathogenesis-related genes and a more marked hypersensitive response compared to afternoon or evening inoculations. This effect has been attributed to longer light exposure after the interaction with the pathogen in plants inoculated earlier. Finally, several reports suggest that some aspects of plant defense responses present circadian rhythm-associated controls. For example, several plant defense-related genes have an expression pattern correlated with the circadian pattern for stomata closure. Moreover, plants deficient in circadian regulation have impaired defense responses (9). Furthermore, Yang *et al.* demonstrated a diurnal change in the resistance of tomato plants to *Pseudomonas syringae* pv. *tomato* DC3000 (Psto), showing the greatest susceptibility before midnight. They also showed that red light (200 μE m^{-2} s^{-1}) treatment enhanced plant resistance. The transcriptome profiling analysis by RNA-seq revealed that SA-mediated signaling pathways, a cellulose synthase and a reduced redox homeostasis are involved in red light-induced resistance to Psto (16).

LIGHT REGULATES THE VIRULENCE IN PHYTOPATHOGENIC BACTERIA

Considering plant pathogens, genes encoding LOV and BLUF photoreceptors are present in bacteria belonging to the genus *Xanthomonas*, *Pseudomonas* and *Ralstonia* (4).

Table 1. Plant-bacterial pathogen interactions systems best studied.

Bacteria	Disease	Photosensory proteins	Processes affected by light/photoreceptors
Xanthomonas citri subsp. *citri* (Xcc)	Citrus canker	LOV BLUF BLUF PHY	Motility, adhesion, biofilm formation, oxidative stress resistance, exopolysaccharide production, symptoms development (20)
Xanthomonas campestris pv. *campestris* (Xcca)	Crucifer black rot	LOV PHY	Exopolysaccharide production, biofilm formation, symptoms development (32)
Pseudomonas syringae pv. *tomato DC3000* (Psto)	Bacterial speck	LOV PHY PHY	Motility, attachment, biofilm formation, exopolysaccharide production, biosurfactant production, type II secretion system, symptoms development (37,41,50, L. Moyano, A. Carrau, S. Petrocelli, I. Kraiselburd, W. Gärtner, E. G. Orellano, unpublished)
Pseudomonas syringae pv. *syringae* (Pss)	Bacterial brown spot	LOV PHY PHY	Motility (51)
Pseudomonas cichorii JBC1	Leaf spot, Midrib rot, Stem necrosis	LOV PHY PHY	Symptoms development (52)
Agrobacterium tumefaciens	Crown gall	Cryptochrome/Photolyase PHY PHY	Motility, attachment, symptoms development (56)

Light regulation and photoreceptor proteins in bacteria of the *Xanthomonas* genus

Xanthomonas citri subsp. *citri* (Xcc) is the bacterium responsible for citrus canker, a disease that affects all commercial varieties of *Citrus*. Bacterial infections that occur early in fruit development result in serious production losses. Late infections only result in scattered necrotic corky lesions on leaves, stems and fruits; however, these fruits become unacceptable for international commercialization (17,18). Xcc presents multiple virulence factors involved in host colonization, plant immune response evasion and disease development. These include type II and type III secretion systems, responsible for secreting enzymes for the degradation of plant cell components and for injecting of virulence effectors inside the host cells, respectively, and other surface structures important for plant colonization such as adhesins, lipopolysaccharides and exopolysaccharides (19). The Xcc genome sequence includes four genes encoding putative photoreceptors: a phytochrome, two BLUF proteins and a LOV protein (20).

The LOV protein of Xcc (Xcc-LOV) presents an N-terminal LOV domain associated with a C-terminal histidine kinase (HK) domain and a response regulator (RR) domain (hybrid HK-RR). The amino acid sequence of Xcc-LOV presents all the essential amino acids for LOV photochemistry, including the characteristic GXNCRFLQ motif containing the conserved cysteine residue involved in the covalent adduct formation upon blue light absorption (C76 in Xcc) (21,22). In fact, Xcc-LOV is a legitimate photoreceptor capable of absorbing blue light and initiates a canonical LOV photocycle (20). In this protein, photochemistry does not take place in C76 mutations; confirming the involvement of this residue in the generation of the covalent photoadduct essential for photochemistry (22). The mechanism by which the Xcc-LOV photoreceptor is activated upon an incoming blue light photon to start a signal transduction cascade has been elucidated by a complete spectroscopic analysis. This study demonstrated that upon photoconversion, the Xcc-LOV protein does not undergo significant conformational changes, as indicated by unaffected fluorescence spectra of tryptophan and tyrosine residues and fluorescence anisotropy. Moreover, laser-induced optoacoustic spectroscopy (LIOAS) studies revealed that, unlike previously characterized LOV proteins, the photoconversion of Xcc-LOV does not involve a volume contraction within the observation time window. However, as the back-conversion into the dark state is, in fact, accompanied by a volume expansion, it can be assumed that the volume contraction associated with adduct formation in the wild-type falls at the limit of the time window for LIOAS or its absolute value is very small. On the other hand, the Xcc-LOV protein exhibits quantum yields for triplet ($\Phi T = 0.68$) and photoadduct ($\Phi_{390} = 0.46$) formation as well as the lifetime for triplet decay ($\tau_T = 2.4$–2.8 μs) similar to other described bacterial LOV proteins including those from *Pseudomonas syringae* pv. *tomato* and *Bacillus subtilis* (22).

The light state of the Xcc-LOV protein is the active (signaling) state, probably initiating a signal transduction cascade involved in a physiological response. This was demonstrated by a radioactivity-based enzyme function assay which revealed a light-induced upregulation of the kinase function of Xcc-LOV (22). Regarding the role of the Xcc-LOV in the bacterial physiology, this protein modulates bacterial features directly associated with the Xcc ability to colonize host plants, such as motility, adhesion, biofilm formation and oxidative stress resistance, by means of the synthesis of flagellum, exopolysaccharides and

adhesins. Moreover, disease development during the bacterial infection of orange trees depends on the functionality of this protein, as considerably different symptoms developed in plants infected with a *lov*-mutant strain of Xcc than those developed upon Xcc wild-type infection, with a higher degree of tissue necrosis (20). An expression profiling of *Citrus sinensis* leaves revealed that genes directly involved in plant defense are upregulated upon infection with a *lov*-mutant strain. The principal biological processes differentially affected by treatments include photosynthesis, sucrose catabolism, secondary metabolism and defense response. Moreover, leaves inoculated with the *lov*-mutant strain present a more pronounced decrease in photosynthesis, a higher lignin accumulation and tissues integrity are more severely affected compared to wild-type Xcc-inoculated leaves (23). Furthermore, a qualitative and quantitative evaluation of reactive oxygen species in *C. sinensis* shows a more marked oxidative burst in plants inoculated with the *lov*-mutant strain of Xcc (I. Kraiselburd, L. Moyano, S. Petrocelli, E. G. Orellano, unpublished) (Fig. 1).

Based on its lifestyle, Xcc is classified as a hemibiotrophic pathogen, which relies on metabolites derived from plant cells to grow. Therefore, maintaining plant photosynthesis and the integrity of host plant tissues favors bacterial establishment and survival. In fact, in Xcc, bacterial modulation of host tissue damage is an important mechanism for surviving in the host long enough to develop the disease and to spread to neighboring tissues (24). Moreover, although the initiation of plant immune responses and the biosynthesis of defense compounds are energy-demanding processes, plant photosynthesis is negatively regulated upon pathogen attack to prioritize the metabolic processes involved in the production of defense compounds (25). The high representation of upregulated genes related to plant defense mechanisms against pathogen attack in tissues inoculated with the *lov*-mutant strain of Xcc, as well as with the enhanced biochemical and structural host tissue alterations, shows a stronger host response upon infection with this strain. These results suggest that Xcc-LOV participates in a bacterial strategy to counteract plant defense responses and to maintain plant energetic metabolism and integrity, thus favoring bacterial survival in host tissues and disease development (plants were grown in a greenhouse with a photoperiod of 16 h light = 150 μE m^{-2} s^{-1}, and 8 h dark at a temperature of 25 °C and 80% humidity) (23).

In the case of Xcc BLUF proteins, preliminary data show that the protein encoded by the *bluf3278* gene is involved in the modulation of different physiological features such as swimming and swarming motility, exopolysaccharide and biosurfactant production and adhesion to orange leaves. Furthermore, this gene participates in the regulation of the interaction between Xcc and the host plant (A. Carrau, J. Tano, L. Moyano, S. Petrocelli, L. M. Moreira, J. Setubal, E. G. Orellano, unpublished). The physiological role of bacterial BLUF proteins is still largely unexplored, and this information represents the first evidence of a functional BLUF photoreceptor in a bacterial plant pathogen.

The related bacterium *Xanthomonas campestris* pv. *campestris* (Xcca) is responsible for crucifer black rot, a disease affecting all cultivated varieties of brassicas worldwide. Infections by Xcca can occur at any stage of the plant life cycle and typical symptoms are v-shaped chlorotic necrotic lesions extending out from the leaf margins and blackening of vascular tissues (26). This bacterium presents several proteins potentially involved in blue, tricolor (blue, red and far-red) or red/far-red light signaling, belonging to three functional groups: HKs, GGDEF proteins and hybrid HKs.
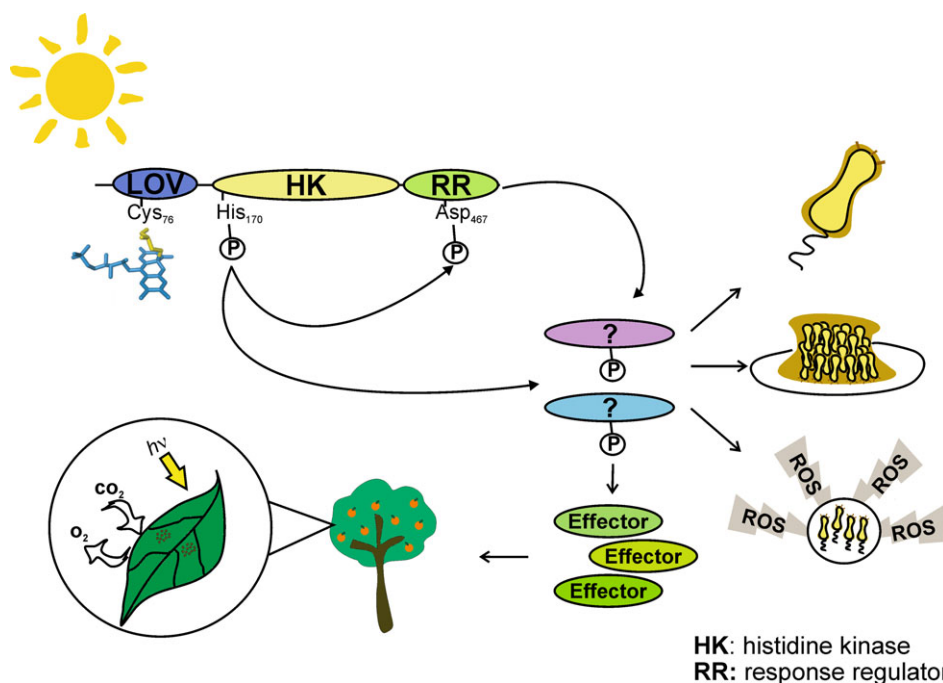
Figure 1. Physiological responses of the molecular activation of the LOV protein from *Xanthomonas citri* subsp. *citri* (Xcc), the bacterium responsible for citrus canker. Xcc-LOV is a blue light sensing, hybrid histidine kinase protein, which presents a canonical LOV photochemistry involving the blue light-induced formation of a covalent cysteine-chromophore (flavin) adduct, preceded by a transient flavin triplet state and followed by thermal conversion to the dark state. Xcc-LOV presents a blue light-induced kinase activity, initiating a still not described transduction cascade involved in regulating the synthesis of flagellum, exopolysaccharides and adhesins, thus modulating bacterial physiology including motility, adhesion, biofilm formation and oxidative stress resistance. Moreover, Xcc-LOV is involved in regulation of the synthesis of bacterial virulence effectors for maintaining plant energetic metabolism and counteracting plant defense (19,22).

Mao *et al.* demonstrated that these proteins modulate growth, motility and virulence of Xcca and are regulated both by the light quality (wavelength) and quantity (intensity) (27). Bacterial growth is modulated by exposure to red (4.30 mW cm^{-2}) and far-red light (3.36 mW cm^{-2}), this effect being dependent on two HKs, two hybrid HKs and three GGDEF proteins. White (12000 lux) and blue light (763 μW cm^{-2}) has a stronger influence on the behavior of Xcca, with three HKs, four GGDEF proteins and four hybrid HK putative involved in the signaling process in response to these radiations (27). Considering blue light in particular, a deletion mutant in an HK protein exhibits an impaired growth under this wavelength, in contrast to what was observed in Xcc, where light does not influence bacterial growth. The influence on bacterial virulence has been established for four light-sensing proteins, three of which present GGDEF domains, confirming that the virulence of Xcca is modulated in response to lighting conditions (27).

Xcca has a single bacteriophytochrome. The crystal structure bearing a full-length BphP with a canonical structure PAS–GAF–PHY photosensory module C-terminally linked to a PAS output domain was recently published (28). In addition, Otero *et al.* demonstrated that the phytochrome of Xcca acts as a bathy-like phytochromes (28,29). These kinds of phytochromes have a longer wavelength Pfr form than the ground state (30,31). The specific functions of the Xcca-phytochrome have been recently described. In this bacterium, the bacteriophytochrome downregulates exopolysaccharide production and attachment in response to light (15 μE m^{-2} s^{-1}). Also, a phytochrome-mutant strain showed enhanced virulence. Moreover, the mutant strain showed an alteration in the stomata aperture regulation and callose deposition (32). Furthermore, Bonomi *et al.* demonstrated

that the far-red light (733 nm; 0.7 W LEDs at a 15 cm distance) or the Xcca-phytochrome overexpression produces the inhibitions of several virulence factors in this bacterium (32).

Effects of light and photosensory proteins on the physiology and virulence of *Pseudomonas* plant pathogenic bacteria

Pseudomonas syringae pv. *tomato* DC 3000 (Psto) bacterium is the causal agent of bacterial speck in tomatoes and other plants including *A. thaliana*. In the first step of infection, Psto develops an epiphytic phase on the plant surface. Then, the bacterium enters into the apoplast through natural openings and wounds and produces the disease symptoms. Bacterial speck is characterized by the development of necrotic symptoms in leaves, stems and fruits (33,34). The Psto genome was completely sequenced (35), and it presents a LOV-HK-RR protein (PSPTO_LOV) with a light-regulated HK activity (36) and two bacteriophytochromes (PSPTO_PHY1 and PSPTO_PHY2), whose activity is triggered by red light (7). The Psto-LOV protein presents a canonical LOV photochemistry, with an absorbance spectrum typical for oxidized flavin species and fluorescence emission in the dark, with the appearance of a species absorbing maximally at 390 nm and loss of flavin fluorescence upon blue light irradiation, indicating the formation of a covalent flavin-protein photoadduct (36). This adduct slowly reverts to the dark state with a lifetime of 5650 s. LIOAS evaluation of this protein revealed similar properties to those observed in other LOV proteins, such as Xcc-LOV. Transient species are formed within 20 ns corresponding to the FMN triplet state, with a small volume contraction. Then, triplet decays with a lifetime of 1.5 μs generating the covalent photoadduct. The quantum yields of triplet and adduct formation are consistent with the values

reported for most LOV proteins; however, unlike other described LOV proteins, adduct formation does not result in a further contraction, but rather in a small expansion (36). Río-Álvarez *et al.* demonstrated that while light does not affect bacterial growth in liquid and solid media, Psto exhibits an inhibition of its typical swarming motility when grown under white (70 μE m^{-2} s^{-1}) or blue light (20 μE m^{-2} s^{-1}). This effect is a consequence of a downregulation of the expression of genes involved in the synthesis of bacterial flagella and in the regulation of flagellar functions. However, bacterial growth under red light (20 μE m^{-2} s^{-1}) and darkness favors bacterial motility, showing the participation of the Psto-LOV protein in the inhibition of this process. In addition, bacterial attachment and biofilm formation in the leaves of host plants are favored under blue light. This is thought to be the result of an upregulation of genes involved in the alginate biosynthesis, an exopolysaccharide involved in adhesion and resistance mechanisms during the epiphytic stage. Based on these effects, the authors suggest that blue light provides a signal for the bacterial switch to a nonmotile attached lifestyle (37). On the other hand, light is involved in the regulation of bacterial virulence in Psto, because bacterial populations and disease symptoms developed in tomato and *A. thaliana* plants are significantly reduced when bacteria are grown under blue light conditions. Bacterial growth in red light causes the opposite effect, increasing the disease symptoms compared to the treatment with bacteria grown in the darkness (37). The reduced virulence of Psto under blue light conditions is thought to be associated with the Psto-LOV-dependent effect of this radiation on bacterial flagella, as these structures are essential for the initiation of bacterial infection (38). In a Psto-LOV-mutant strain, bacterial virulence is reduced in comparison with the wild-type strain despite the light conditions and it is not affected by blue or white light. Moreover, the opposite effect is observed under red light, suggesting that bacteriophytochromes also have a role in the virulence of this bacterium (37). LOV protein in Psto is encoded by a gene present in a pathogenicity island (LOV domain gene genomic island or LOV–GI), which also encodes genes involved in circadian timing (a gene homologous to *lab*A from *Synechococcus*) (39), light responses (40) and putative virulence factors (41). Despite not affecting bacterial growth in rich media, Psto-LOV protein seems to be involved in the light-dependent inhibition of the growth of Psto in minimum media. Consistent with these facts, Psto-LOV protein has been shown to be a master negative regulator of gene expression in this plant pathogen. Psto-LOV is involved in the light-dependent downregulation of the expression of genes encoding the principal sigma factors highly expressed during bacterial exponential growth and alternative sigma factors involved in general stress response, secondary metabolite production, nutrient-scavenging stress response and production of type III secretion system and type III virulence effectors (42,43). As established by Río-Álvarez *et al.* and Moriconi *et al.* repression of these genes by Psto-LOV protein can account for the reduced bacterial proliferation in *A. thaliana* plants inoculated with Psto grown under white and blue light conditions, not only by an impaired bacterial motility and thus inability of bacterial infection, as previously mentioned, but also as a result of a reduced bacterial replication once inside the plant apoplast (37,41). Moreover, the Psto-LOV-directed repression of type III secretion system and effectors probably results in the reduced virulence reported for Psto in the light (37). In fact, Moriconi *et al.* demonstrated that this effect is the result of an impaired bacterial invasiveness in leaf tissues, that is the capacity of the pathogen to grow and spread within the host (41). Psto is a hemibiotrophic pathogen that grows epiphytically on plant surfaces without causing disease symptoms until it enters the plant leaf apoplast, where it multiplies by exploiting live host cells. This bacterium also survives as a saprophyte in the soil (44–47). Moriconi *et al.* also suggested that while the lack of effect of Psto-LOV in the darkness may favor bacterial survival in saprophytic conditions, reduced bacterial proliferation in the light can reduce the damage in the host tissue, thus optimizing the bacterial nutritional resources in order to allow the dispersal to new hosts (41). Phytochromes of Psto are biliverdin IXα (BV)-binding phytochromes. *Psto*bphP1 is arranged in an operon with a heme oxygenase (*Psto*BphO)-encoding gene (*Psto*bphO), whereas *Psto*bphP2 is flanked downstream by a gene encoding a CheY-type response regulator (7). Concerning the role of the bacteriophytochromes in the physiology of Psto, these proteins modulate several features related to the epiphytic phase. The ability to adhere to biotic surfaces and biofilm development are regulated by phytochromes, as these proteins have been shown to be necessary for a successful bacterial attachment. The production of biosurfactants, which is another feature related to the epiphytic life of bacteria (48), is markedly reduced in Δ*bphP1* and Δ*bphP2* strains, indicating that phytochromes of Psto have a positive regulation of these features. In agreement with this result, epiphytic fitness assays showed that tomato leaves inoculated with mutant strains presented less typical bacterial speck symptoms. Swarming motility has an essential role in the colonization of natural environments, the flagellum being the most important requirement for this kind of motility (49). Migration zones of Psto strains exposed to darkness are higher than those observed when bacteria are exposed to white (20 μE m^{-2} s^{-1}) and red light (40 μE m^{-2} s^{-1}) conditions. In red light, swarming motility is decreases but is not completely repressed. Moreover, Δ*bphP1* and Δ*bphP2* mutants migrate further than the wild-type strain in every light condition. The examination of the flagella from swarming plates by transmission electron microscopy (TEM) showed that, in agreement with the bacterial swarming observation, the wild-type strain in darkness presented thicker flagella than in white and red light. On the other hand, the wild-type strain grown under red light presented a fewer number of flagella, while both mutant strains presented a greater number of flagella than the wild type in all lighting conditions. The biological significance of different light treatments and the phytochromes of Psto in the pathogenesis process were determined by studying the ability of this bacterium to develop disease symptoms in tomato plants under different light conditions. Leaves inoculated with wild-type and Δ*bphP1* strains in tomato plants exposed to white light showed typical bacterial speck lesions, while Δ*bphP2*-inoculated leaves produced highly necrotic lesions. In red light, no lesions were observed after the inoculation with all the Psto strains. On the other hand, when inoculated leaves were maintained in dark conditions, the mutant strains showed a higher necrotic lesion than the wild-type strain. This pattern of infection, which depends on the different light conditions, is also observed in epiphytic fitness assays suggesting that the light is an important factor both in Psto pathogenicity and in the plant response (L. Moyano, A. Carrau, S. Petrocelli, I. Kraiselburd, W. Gärtner, E. G. Orellano, unpublished) (Fig. 2).

Ricci *et al.* studied some Psto strains lacking genes in LOV protein (Δ*lov*A and Δ*lov*B), in bacteriophytochrome 1 (Δ*BphP1*) and in heme oxygenase (Δ*HO*-A and Δ*HO*-B) and under different continuous light qualities or in the dark. The authors showed that *Pst*LOV and *Pst*BphP1 downregulate bacterial growth under white light, red (6 μE m^{-2} s^{-1}) and far-red (17 μE m^{-2} s^{-1}) and in

darkness. In addition, under blue light (40 µE m^{-2} s^{-1}), the mutant strains are not able to grow in liquid culture suggesting that the simultaneous presence of *Pst*LOV, *Pst*HO and *Pst*BphP1 is essential to guarantee growth under blue light. On the other hand, Ricci *et al.* inferred that these bacterial photoreceptors downregulated the infectivity in *A. thaliana* host plants (50).

Another bacterium from the *Pseudomonas* genus is the bean pathogen *Pseudomonas syringae* pv. *syringae* (Pss). This bacterium also presents two bacteriophytochromes, BphP1 and BphP2, and a LOV-HK protein (51). In Pss, swarming motility is modulated by light like Psto and Xcc. Wu *et al.* demonstrated that in Pss, LOV-HK protein behaves as a positive regulator of swarming motility in response to blue light (5 µE m^{-2} s^{-1}) and that BphP1 represses swarming motility in response to red (10 µE m^{-2} s^{-1}) and far-red light (0.8 µE m^{-2} s^{-1}), both being the red-light-absorbing (Pr) and a far-red-light-absorbing (Pfr) forms responsible for the inhibition phenotype. Interestingly, BphP1 is also involved in the response of this bacterium to blue light, acting as a repressor of swarming motility downstream of LOV-HK signal transduction pathway. On the other hand, BphP2 seems to have no role in bacterial motility through light modulation (51). In this way, in Pss, BphP1 and LOV-HK proteins appear to constitute an integrated signaling network where BphP1 responds to blue light as well as red and far-red light and LOV-HK suppresses BphP1-mediated blue light signaling. Wu *et al.* suggested that this plant pathogen, sensing distinct aspects of light quality, probably aims at favoring bacterial epiphytic survival and host colonization (51).

Nagrendran *et al.* studied the influence of light on the interaction between another *Pseudomonas* strain, *Pseudomonas cichorii* JBC1 and tomato plants. This bacterium is responsible for the leaf spots on soybean, mid-rib rot on lettuce and leaf spot and stem necrosis in tomato. Like Psto, *P. cichorii* does not present significant changes in the growth rates when it is grown under different lighting. Tomato seedlings inoculated with *P. cichorii* grown under white light (120 µE m^{-2} s^{-1}) or in the dark show severe necrotic lesions. However, disease symptoms are significantly lower when seedlings are exposed to red (120 µE m^{-2} s^{-1}) or green light (120 µE m^{-2} s^{-1}). Assays performed with mature tomato plants showed the same phenotype. According to this result, the analysis of the expression of defense-related genes in *P. cichorii*-infected tomato seedlings grown under red and green light showed that these genes are upregulated in these conditions. Furthermore, lighting did not influence swarming motility and biofilm formation in this bacterium. Nagendran *et al.* showed that tomato plants grown under red and green light suppress disease development by regulating defense-related gene expression (52).

Light-regulated processes in the soil bacterium *Agrobacterium tumefaciens*

Finally, light effects on the physiology and virulence of the plant pathogen *Agrobacterium tumefaciens* have been reported. This bacterium is the causal agent of crown gall disease in over 140 plant species, including several economically important crops. Disease symptoms include the formation of tumor-like swellings (galls) at the crown of the plant, just above soil level as a consequence of the insertion of a small segment of DNA from a bacterial plasmid into the plant cell, which is incorporated at a semi-
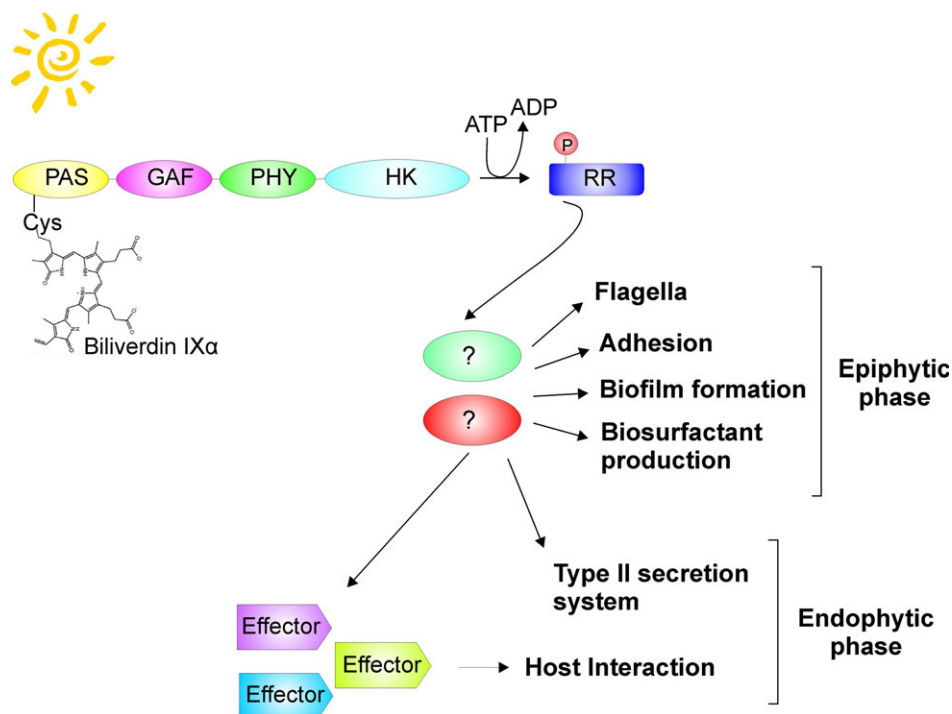

Figure 2. Physiological responses of the molecular activation of the phytochrome proteins from *Pseudomonas syringe* pv. *tomato* DC3000 (Psto), the bacterium responsible for bacterial speck. Bacteriophytochromes (Bphs) use a biliverdin IXα as a chromophore bound via a thioether linkage. These photoreceptors show photochromicity, the canonical phytochromes, in particular, photoconvert between red-absorbing (Pr) and far-red-absorbing (Pfr) states. Psto phytochromes are involved in the regulation of some features related to the epiphytic phase including swarming motility through synthesis of flagellum, adhesion, biofilm formation and biosurfactant production. In addition, phytochromes are involved in regulation of endophytic phase of Psto plant colonization. The transduction cascade is still not described.

random location into the plant genome (53). *Agrobacterium tumefaciens* presents two phytochrome genes and a gene encoding a cryptochrome/photolyase-type protein (54,55). In *A. tumefaciens*, white light (150 µE m^{-2} s^{-1}) represses the expression of flagellum genes, producing a reduction in bacterial motility. Also, bacteria grown in darkness adhere better to the plant tissue and are more virulent (56). Oberpichler *et al.* suggested that, in the dark, *A. tumefaciens* exhibits an increased virulence to optimize the colonization and the infection of host plants when plant defenses are less efficient. Alternatively, the authors suggested that an increased motility in the dark could be part of a mechanism to address the bacteria to the base of the plant, where tumors are preferentially formed (56). The mechanism responsible for these light responses in *A. tumefaciens* remains unknown and it is still unclear which photoreceptor is responsible for the light regulation of bacterial virulence (56). Both phytochromes of *A. tumefaciens*, *At*BphP1 and *At*BphP2, have been extensively characterized. *At*BphP1 behaves as a typical BphP; it assembles with biliverdin IXα to generate a Pr ground state that has high histidine autophosphorylation activity. After photoconversion to Pfr by red light absorption, this activity is immediately repressed but slowly restored as Pfr reverts back to Pr in the dark. In contrast, *At*BphP2 assembles with biliverdin IXα and first generates a transient Pr-like intermediate, which then transforms nonphotochemically into a stable Pfr form with high histidine kinase activity. After photoconversion to Pr by far-red light absorption, this kinase activity drops but is quickly restored as the unstable Pr form rapidly reverts to Pfr (54,57). Karniol and Vierstra suggest that *A. tumefaciens* uses these mutually opposing BphPs to detect its location within the soil strata sensing the red light/far-red light ratio (30).

CONCLUDING REMARKS

In conclusion, the reported effects of light on phytopathogenic bacteria show that the light environment constitutes an important cue that influences the outcome of plant–pathogen interactions, not only by affecting plant defense responses but also by modulating the virulence of plant pathogens. In this way, phytopathogens appear to have developed mechanisms to employ an environmental factor that is essential for the activation of plant defense responses, as a regulator for modulating its virulence in response to its host.

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AUTHOR BIOGRAPHIES

Ivana Kraiselburd is a Marie Skłodowoska-Curie postdoc fellow at the Biofilm Centre, University Duisburg-Essen. She obtained her Ph.D. degree from the University of Rosario for her work at the Institute of Molecular and Cell Biology (IBR), where she studied photoreceptors from plant pathogens and their effect in bacterial virulence. She performed a postdoctoral training at IBR studying adaptation processes in phytopathogens including phenolic compounds detoxification. Through international collaboration projects and exchange fellowships, she was invited researcher at the Valencian Institute of Agricultural Research, Spain, the University of São Paolo, Brazil, and the Max Planck Institute for Chemical Energy Conversion, Germany.

Laura Moyano is a Ph.D. student at the University of Rosario, Argentina. She works at the Molecular Genetics and Functional Genomics of Plant-Microorganism Interactions Laboratory in the Institute of Molecular and Cell Biology from Rosario (IBR-CONICET-UNR). Her thesis project is about the role of photoreceptor proteins, specifically phytochromes and BLUF proteins, during plant–pathogen interaction.

Analía Carrau is a Biotechnologist graduated from the University of Rosario, Argentina. She is a Ph.D. student at the University of Rosario who works at the Molecular Genetics and Functional Genomics of the Plant-Microorganism Interaction Laboratory in the Institute of Molecular and Cell Biology from Rosario (IBR-CONICET-UNR) in a project about bacterial photoreceptors and their role and plant–pathogen interaction.

Josefina Tano is a microbiologist graduated from the National University of Río Cuarto, Argentina. Today, she is a Ph.D. student in the National University of Rosario and has a fellow from CONICET. She is currently at the Institute of Molecular and Cell Biology from Rosario (IBR-CONICET-UNR), working in a project about the role of light in *Xanthomonas citri* subsp. *citri* and *Ralstonia solanacearum*.

Elena G. Orellano is graduated in Biochemistry and Ph.D. in Biological Sciences from the University of Rosario, Argentina. Currently she is a professor of Molecular Biology in the Department of Biological Sciences at the same university. The main focus of her research work is to understand the molecular basis of bacterial pathogenicity during plant–pathogen interactions, using as models the phytopathogens *Xanthomonas citri* subsp. *citri* and *Pseudomonas syringae* pv. *tomato*. One of the subjects of her studies is the characterization of blue light and red light bacterial photoreceptors and their involvement in the bacterial physiology and in the pathogenicity process.