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Illuminating the *Arabidopsis* circadian epigenome: Dynamics of histone acetylation and deacetylation

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Abstract

The circadian clock generates rhythms in biological processes including plant development and metabolism. Light synchronizes the circadian clock with the day and night cycle and also triggers developmental transitions such as germination, or flowering. The circadian and light signaling pathways are closely interconnected and understanding their mechanisms of action and regulation requires the integration of both pathways in their complexity. Here, we provide a glimpse into how chromatin remodeling lies at the interface of the circadian and light signaling regulation. We focus on histone acetylation/deacetylation and the generation of permissive or repressive states for transcription. Several chromatin remodelers intervene in both pathways, suggesting that interaction with specific transcription factors might specify the proper timing or light-dependent responses. Deciphering the repertoire of chromatin remodelers and their interacting transcription factors will provide a view on the circadian and light-dependent epigenetic landscape amenable for mechanistic studies and timely regulation of transcription in plants.

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Circadian clock, Light signaling, Histone acetylation, Histone deacetylation, *Arabidopsis thaliana*.

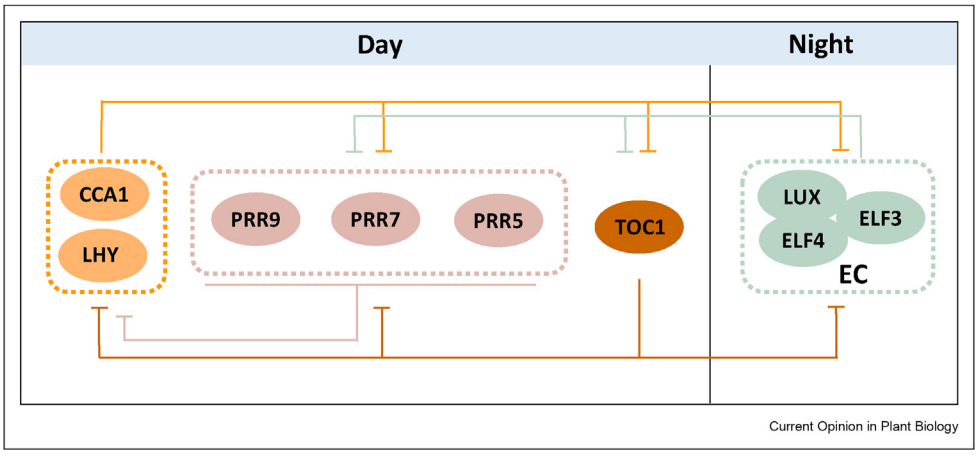
Introduction

Plants, as many other organisms, use a cellular mechanism or circadian clock to coordinate biological processes in synchrony with the environmental time. The main output of the circadian function is the generation of 24-h biological rhythms precisely timed to occur at the most favorable daily or seasonal time [1]. Consistently, the circadian function has been proposed to improve fitness and survival, providing an adaptive advantage [2]. The importance of the circadian function in plants is also manifested by the plethora of processes regulated by the clock, including among others, physiological responses, metabolic homeostasis or cellular growth and development [1–4]. Generation of rhythms relies on the proper perception of environmental cues that set the time-of-day [5], and the delicate cross-regulation among oscillator components that ultimately generate rhythmic oscillations in the output processes regulated by the clock [1].

The main oscillator components in *Arabidopsis thaliana* display sequential peaks of expression and activity during the day or night (Figure 1). For instance, clock components acting during the day include the single MYB transcription factors CIRCADIAN CLOCK ASSOCIATED1 (CCA1) and LATE ELONGATED HYPOCOTYL (LHY) and the morning-expressed Pseudo-Response Regulators PRR9 and PRR7 [1,6], which act mainly as transcriptional repressors. Other morning-expressed components that function as activators include the REVEILLE (RVE) protein family and NIGHT LIGHT-INDUCIBLE AND CLOCK-REGULATED GENES (LNKs). Later during the day and close to dusk, PRR5 and TIMING OF CAB EXPRESSION1 (TOC1/PRR1) exert a repressive function [1,6] that is temporally followed by the activity of EARLY FLOWERING 3 (ELF3), ELF4 and LUX ARRHYTHMO/PHYTOCLOCK1 (LUX/PCL1), which assemble into the Evening Complex (EC) to repress gene expression during the night [7]. Overall, multiple regulatory mechanisms are responsible for the regulation of rhythmic gene and protein expression and activity. The mechanisms pervade different stages of transcriptional and translational regulation [6,8,9]. The past years has also seen an increase in our understanding on the connection between the circadian function and changes in chromatin remodeling [10].

Post-translational modifications of histones are one of the well-studied mechanisms associated with transcriptional regulation [11]. The link of these modifications with

Figure 1



Simplified transcriptional regulatory network at the core of the Arabidopsis circadian clock. The expression and function of the main oscillator components occur at specific times during the day and night. Most of the components act as repressors of oscillator gene expression (red lines) although some components have been described as activators (blue lines). Tripartite protein complex known as the Evening Complex (EC) is delimited by the dotted line. Please consult the text for further details.

transcriptional regulation relies on particular histone modifications favoring chromatin conformation changes that result in permissive or repressive chromatin states and thus affecting the transcriptional machinery accessibility and transcription factor binding [12]. Histone acetylation is one of the well-studied histone modifications correlated with gene activation. Acetylation relies on the activities of histone acetyltransferases (HATs) that ensure chromatin commitment for inducible gene activation. The HAT activities are dynamically counterbalanced by histone deacetylases (HDACs), which favor deacetylation and gene repression. The correlation between rhythmic changes in gene expression with oscillatory chromatin conformations and histone acetylation has been previously established [10]. Several studies have also identified the chromatin-related factors contributing to these rhythmic oscillations [10].

Light is one of the main environmental cues responsible for resetting the clock every day [5]. Light also regulates many essential aspects of plant growth and development [13]. For instance, upon light perception, photoreceptors such as PHYTOCHROMES and CRYPTOCHROMES initiate complex molecular cascades to reprogram seedlings for photomorphogenesis [14]. Conversely, under dark conditions, seedlings undergo skotomorphogenesis characterized by closed cotyledons, and elongated hypocotyls. The CONSTITUTIVE PHOTOMORPHOGENIC1/DEETIOLATED/FUSCA (COP1/DET/FUS) E3 ligase complex [15] act as a core repressor of photoreceptor signaling cascade by promoting in the dark, the proteasome degradation of many key transcription factors, including among others ELONGATED HYPOCOTYL 5 (HY5) or FAR-RED ELONGATED HYPOCOTYL3 (FHY3) [16]. Thus, an intricate balance between photoreceptors and COP1 signaling cascades under light and dark conditions defines the photomorphogenesis or skotomorphogenesis reprogramming, respectively.

The circadian system and light signaling pathways regulate each other, shared molecular components and regulatory mechanisms. One of the shared regulatory mechanisms is the changes in chromatin conformation and its correlation with transcriptional regulation [10,17]. In this review, we provide a glimpse of the commonalities and divergences of the epigenetic regulation of clock- and light-related signaling pathways, with particular emphasis on histone acetylation and deacetylation. We briefly describe circadian and light-mediated regulatory changes in chromatin status and the chromatin remodelers involved in such regulations. Lessons learnt in one pathway can lead the way for similar discoveries in the other.

Circadian and light-dependent changes of histone acetylation

Since the first study, nearly 15 years ago, showing that rhythmic changes in Histone 3 (H3) acetylation at the promoter of a clock gene correlated with the transcriptional rhythms of clock gene expression [18], many other oscillator genes have been shown to be rhythmically decorated with different chromatin marks [10]. The generation of transcriptional rhythms in steady-state and nascent RNAs [19], relies on a complex interplay between chromatin remodelers and oscillator components. Rhythmic changes in chromatin rely on the circadian regulation of the expression chromatin-related

factors [20], and/or in the direct interaction of oscillator components with chromatin-related factors. These mechanisms ultimately allow the rhythmic recruitment of the chromatin-related factors to the clock loci [10].

The study showing that the raising phase of *TOC1* oscillatory waveform correlated with rhythms in H3 acetylation at the *TOC1* promoter [18] was later followed by evidence of oscillatory changes in histone acetylation at the promoters of other oscillator genes [21–23]. The two morning-expressed single MYB transcription factors CCA1 and RVE8 were found to perform antagonistic functions shaping the circadian waveform of histone acetylation at the *TOC1* promoter. While CCA1 repressed *TOC1* and favored histone deacetylation [18], RVE8 activated *TOC1* expression by enhancing to histone acetylation [24]. The transcriptionally permissive chromatin conformations favored by RVE8 correlated with the rhythmic recruitment of the transcriptional machinery through interaction with the RNA Polymerase II and the FACT complex [19]. However, the chromatin remodelers involved in histone acetylation, working in conjunction with RVE8 remain to be identified.

Changing light conditions also modulate the pattern of histone modifications such as acetylation at light-regulated gene loci. This mechanism directly connects light with transcriptional regulation [25,26]. Distinct photoreceptors contribute to the effects of the different light qualities on histone modifications [26]. Notably, the expression of the photoreceptors themselves seems to be regulated by chromatin remodeling. For instance, the up-regulation of the photoreceptor *PHYTOCHROME A* (*PHYA*) is associated with an increased pattern of histone acetylation at the *PHYA* locus [27]. Some of the components involved in the regulation of histone acetylation have been reported, including the master repressors of photomorphogenesis COP1 and DET1, which are involved in the light-dependent control of gene-specific histone modifications [26].

The connection of COP1 and DET1 with the plant circadian clock has been shown in several studies. For example, COP1 directly interacts with the clock component ELF3 to regulate photoperiodic flowering [28]. Other examples include the interaction of DET1 with CCA1 and LHY, which contributes to the transcriptional repressive function of CCA1 and LHY [29]. Furthermore, COP1 SUPPRESSOR 4 (CS4), a suppressor of the *cop1* mutant, also represses CCA1 target genes by directly interacting with CCA1 [30]. Thus, COP1 and DET1 interaction with clock components seems to be important for regulation of clock gene expression and light-dependent clock outputs.

An example of a chromatin-remodeling factor regulating both rhythmic clock gene expression and light-dependent transcriptional regulation is the HISTONE ACETYLTRANSFERASE OF THE TAFII250 FAMILY 2 (HAF2/TAF1). Indeed, a recent study has shown that CCA1 controls the expression of *HAF2*, which activates clock genes expressed close to dusk or during the night [31]. The same chromatin modifier, HAF2 [32–34], is an important activator of light-regulated gene expression (red/far-red and blue light signals) through histone acetylation. Thus, HAF2 regulates both night-expressed clock components and light-regulated genes. Elucidating the molecular components directing HAF2 to the light and clock-related target loci might provide clues into the specificities of this dual regulation.

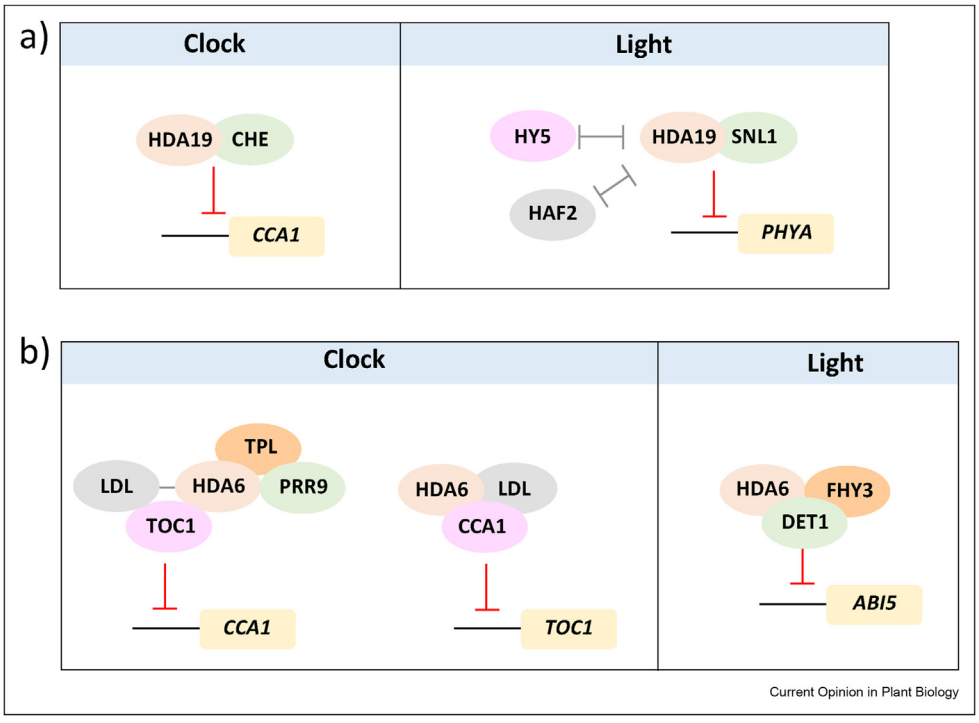
The histone acetyltransferase GENERAL CONTROL NONDEREPRESSIBLE 5 (GCN5) was also proposed to favor acetylation of histone lysine residues and chromatin commitment for light-dependent up-regulation of gene expression [32,33]. Interestingly, GCN5 and HAF2 were both required for sustaining histone acetylation (H3K9, H3K27, and H4K12) on the target promoters, whereas H3K14 acetylation was proposed to rely only on GCN5 [32]. As GCN5 is associated with a large number of gene promoters involved in many different processes [33], it is possible that GCN5 might be involved in the transcriptional regulation of clock genes. If that is the case, GCN5 may directly interact with clock proteins to regulate circadian gene expression.

Recent studies have also shown that the basic helix-loop-helix transcription factors known as PHYTOCHROME-INTERACTING FACTORs (PIFs) contribute to H3 acetylation and removal of the histone variant H2A.Z in response to changes in light quality [35]. The study shows that the epigenetic function of PIFs relies on the interaction with EIN6 ENHANCER, the homolog of the chromatin remodeling complex subunit INO80 Subunit 6 [35]. Notably, the INO80 complex represses the transcription of a central clock gene in *Neurospora crassa* [36]. Thus, future studies could focus on whether the plant INO80 complex also contributes to circadian clock gene expression in plants.

Histone deacetylation and repression of light- and circadian-regulated genes

Several HDACs relate to both the circadian clock and light signaling. For instance, the HISTONE DEACETYLASE 1/HISTONE DEACETYLASE 19 (HD1/HDA19; herein HDA19) (Figure 2a) interacts with the clock-related component CCA1 HIKING EXPEDITION (CHE/TCP21) to regulate the morning-expressed clock gene *CCA1* [37]. HD1/HDA19, together with the SWI-INDEPENDENT3 LIKE (SNL1 and SNL6), also act as a negative regulator of the light signaling pathway by facilitating histone deacetylation of the *PHYTOCHROME A* (*PHYA*) locus [38]. HDA19 regulation of light-dependent gene expression might be achieved by

Figure 2



Examples of shared histone deacetylases related to both the circadian clock and light signaling. a) HDA19 interacts with the clock component CHE to regulate *CCA1* expression, and with several light-related components in the regulation of light signaling pathways. b) HDA6 interacts with clock proteins and light-signaling components to the circadian clock and light signaling pathways. Red lines denote repression. Please consult the text for further details.

counteracting the histone acetylation activities of HAF2 and GCN5 [32]. Light-dependent regulation might also rely on HDA19 antagonistic function with the photomorphogenic transcription factor ELONGATED HYPOCOTYL5 (HY5) [38] (Figure 2a). Whether HDA19 counteracts HAF2 activity in the regulation of clock genes remains to be identified.

Another HDAC, the HISTONE DEACETYLASE-6 (HDA6) forms a protein complex with members of the Groucho/Tup1 protein family, topless/topless-related (TPL/TPR), and with the clock component PRR9 for transcriptional repression of the morning-expressed clock genes *CCA1* and *LHY*, most likely by modulating the pattern of H3 deacetylation [39] (Figure 2b). Molecularly, this regulation appears to be mediated by HDA6 interaction with the LYSINE-SPECIFIC DEMETHYLASE 1 (LSD1)-LIKE 1/2 (LDL1/2). The complex is recruited by TOC1 to the clock-related target loci to repress their expression [40]. In turn, the HDA6-LDL1/2 complex interacts with CCA1 and LHY to repress *TOC1* expression [41]. Thus, HDA6 regulates both morning- and evening-expressed clock genes by interaction with LDL1/2 and with many clock components including PRR9, CCA1, LHY and TOC1 (Figure 2b).

HDA6 has been also connected with light signaling. For instance, natural variation studies identified polymorphic alleles of HDA6 and PHYB involved in light-dependent regulation of chromatin compaction associated with acclimation [42]. HDA6 has been associated with other pathways, such as light-dependent seed germination by carbon monoxide [43] or the light-mediated nitric oxide (NO) signaling [44]. Furthermore, HDA6 is recruited by DET1 and FAR-RED ELONGATED HYPOCOTYL3 (FHY3) to the *ABA INSENSITIVE5* (*ABI5*) locus to contribute to the light- and ABA-dependent control of seedling greening [45] (Figure 2b). Similar to the circadian system, HDA6 regulates light signaling pathways by interaction with an array of different photomorphogenic-related components. Further studies could focus on the possible role of HDA6-LDL1/2 complex in light signaling and its regulated output pathways.

The HISTONE DEACETYLASE 15 (HDA15) is a central hub connecting light signaling and chromatin changes at multiple levels (Figure 3). Indeed, HDA15 interacts with a number of photomorphogenic-related components to regulate the pattern of histone acetylation at the target genes. For example, HDA15 represses gene expression in etiolated seedlings through its

interaction with PHYTOCHROME INTERACTING FACTOR3 (PIF3). Red light represses the binding to the target loci, providing a mechanism whereby PIF3 and HDA15 repress chlorophyll biosynthetic and photosynthetic genes [46]. HDA15 also interacts with PIF1 to regulate the expression of light-responsive genes involved in seed germination [47]. HDA15 also directly interacts with HY5 to repress hypocotyl growth under red and far-red light conditions. The HDA15 and HY5 interaction also regulates H4 acetylation of cell-wall and auxin-related genes [48].

HDA15 also controls HY5 and PIFs through its interaction with COP1. Indeed, HDA15 modulates COP1 repressing function in the regulation of HY5 and PIF3 protein abundance in a light-dependent manner. The authors thus propose that HDA15 positively regulates photomorphogenesis through a post-translational mechanism [49]. It would be interesting to examine whether HDA15 and COP1 are also involved in the regulation of clock protein abundance. HDA15 regulatory function might be also defined by its subcellular localization. Indeed, light and dark conditions control the nucleocytoplasmic shuttling of HDA15: while light favors HDA15 nuclear accumulation, dark conditions trigger HDA15 export out of the nucleus [50].

HDA15 also interacts with the Nuclear Factor-YC (NF-YC) proteins under light conditions, and represses the expression of hypocotyl elongation-related genes by controlling H4 acetylation at their promoters. Under darkness, the HDA15-NF-YC complex dissociates from the promoters leading to increased H4 acetylation and etiolated growth [51]. Interestingly, the clock component TOC1 assembles into a protein complex comprising NF-YB/C, which recruits HDA15 to repress growth-related gene expression [52]. Further studies are required to examine in detail the diurnal changes in HDA15 subcellular localization and its implication in the regulation of evening-expressed clock genes.

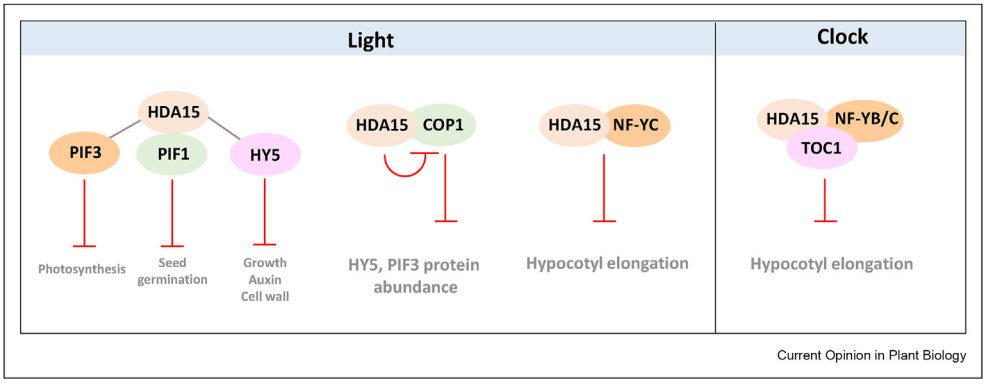
The histone deacetylase (HDA9) also contributes to the repression of many genes by interacting with a wide range of proteins [53] including those involved in light signaling and the circadian system. Indeed, HDA9 interacts with the Evening complex (EC) component EARLY FLOWERING 3 (ELF3) and favors histone deacetylation at the *TOC1* promoter and *TOC1* repression after dusk [54]. HDA9 also functions synergistically with HY5 within the autophagy pathway, and in response to light-to-dark transitions and nitrogen starvation [55]. In addition to its role as a repressor favoring histone deacetylation, HDA9 can also function in transcriptional activation [53].

The circadian clock rhythmically regulates the expression of some chromatin related components [20]. The biological relevance of such oscillation has been demonstrated in studies of the SWI-independent 3/histone deacetylase (Sin3-HDAC) genes *SAP30 FUNCTION-RELATED 1* (*AFR1*), and *AFR2*, which are expressed in the evening. The AFR proteins bind to the *CCA1* and *PRR9* gene promoters and repress their expression during the night by favoring histone deacetylation at their promoters [56]. The MADS transcription factor AGAMOUS-like 15 (AGL15) also interacts with members of the SIN3/HDAC complex providing a mechanism whereby AGL15 represses its target genes [57]. It would be interesting to examine whether AFR1 and AFR2 regulate light signaling pathways by direct interaction of photomorphogenic-related components.

Perspectives

Light signaling modulates chromatin compaction, heterochromatin reorganization and transcriptional reprogramming through processes that are regulated by the

Figure 3



HDA15 lies at the interface of light and clock regulated processes. The interaction of HDA15 with light- and clock-related components in the control of several relevant processes including photosynthesis and growth, among others. Red lines denote repression. Please consult the text for further details.

photoreceptors CRYPTOCHROMES and PHYTOCHROME B [17]. COP1 and DET1 also contribute to a decondensed state of heterochromatin in etiolated cotyledons [17]. The connection of the photoreceptors and COP1/DET1 with the circadian clock place the spotlight into detailed studies on the dynamic changes of chromatin compaction depending on the time-of-day. Furthermore, the results showing that light is able to modulate gene position within the nucleus pave the way for circadian studies using super-resolution microscopy, adapted chromatin profiling or chromosomal conformation capture. Obtaining detailed topological maps of the circadian genome at different times during the day and night will allow a full understanding of the circadian nuclear architecture, higher-order chromatin organization and gene repositioning over the circadian cycle. Plenty of studies are still ahead of us to fully dissect the plant light- and circadian epigenome.

Author contributions

L.X, W.Z. and P.M. wrote the manuscript. All authors accepted the final version.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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