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·特邀综述·

生物钟与光温环境信号互作网络研究进展

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摘要 随着全球气候的急剧变化, 植物生长发育所处的生态环境日益恶劣, 生物钟与光、温受体互作协同传递环境信号并调控下游生长发育的应答机制开始受到科研人员的广泛关注。生物钟作为植物内源计时器, 其核心由多个耦联的转录-翻译反馈环(TTFL)组成, 在转录、转录后、翻译、翻译后和表观遗传层面受到多层级精细调控。这些精密的调控机制保证了生物钟能不断被外界环境信号驯化和重置, 使内源节律与外界环境相匹配, 从而赋予植物优化资源利用和趋向最适生长的能力, 对于指导农作物遗传改良和引种驯化具有重要意义。该综述总结了生物钟核心振荡器的多层级调控机制以及生物钟同源基因在农作物中的分子功能, 详述了生物钟与光、温环境信号通路间的互作网络, 展望了以此为基础的作物分子育种, 为提高农作物的环境适应性和优化改良农艺性状提供了新思路。

关键词 生物钟, 光温信号, TTFL, 环境适应性, 分子育种

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由于地球自转和围绕太阳公转, 地球上的生物必须面临光照、温度和降水等环境因素昼夜性和季节性变化。为感知、预测和适应这些周期性变化的环境因子, 并据此调整生物体内的代谢过程, 地球上的生物进化出一种内源计时机制, 即生物钟(Harmer, 2009)。生物钟分子系统由输入途径、核心振荡器和输出途径3部分组成。从20世纪初至21世纪, 随着植物生物钟核心振荡器转录-翻译反馈环(transcription-translation feedback loop, TTFL)的逐渐建立, 在时间生物学领域开始涌现出大量探索生物钟组分在转录后、翻译后和表观修饰层面的调控机制及生物钟调控下游发育过程的研究, 特别是生物钟基因参与调控农艺性状方面(Xu et al., 2022b)。这些工作不仅进一步丰富和完善了人们对生物钟系统的认识, 也为实现人为调控生物钟周期提供了理论指导和潜在的靶基因。

植物固着生长的特性限制了它们自由躲避环境危害的能力, 因此生物钟精准预测环境变化的能力对其生长发育尤为重要。环境授时因子通过相应的受体或者直接作用于生物钟组分, 使生物钟感知光和温度信号变化, 并诱导生物钟产生与之同步的相位与振幅。生物钟整合环境输入信号后, 进一步协调下游表达及代谢调控网络, 保证生物体生理机能和代谢速率适应外界环境变化, 从而在1天或1年的最适时间完成各个生物学过程, 这对于提高生物体的环境适应能力和存活率至关重要(Steed et al., 2021; Venkat and Muneer, 2022)。

光照和温度是最重要的环境授时因子。首先, “万物生长靠太阳”说明光对植物的生长发育起着决定性作用。其次, 随着全球气候的变化, 低温和高温胁迫频发, 不仅严重影响植物的生长发育和繁殖等各个方面, 而且造成世界范围内农作物产量和品质降低, 因而植物响应温度信号的应答机制备受关注(Ohama et al., 2017; 谢启光和徐小冬, 2024)。近年来的研究表明, 生物钟与光、温环境信号存在复杂的互作网络,

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研究植物感知并启动光、温应答的分子机制，对于引种驯化、扩大纬度适应性及通过分子设计育种提高植物的存活率、改良农艺性状和培育耐低温/高温作物新品种具有重要指导意义。

1 植物生物钟系统

1.1 拟南芥生物钟核心振荡器分子网络研究进展

1.1.1 核心振荡器转录-翻译反馈环

早期的研究表明，核心振荡器由一系列转录抑制因子构成的负-负转录调控环组成，包括CCA1 (CIRCADIAN CLOCK-ASSOCIATED 1)、LHY (LATE ELONGATED HYPOCOTYL)和GI (GIGANTEA)，及晚间复合物组分ELF3 (EARLY FLOWERING 3)、ELF4和LUX (LUX ARRHYTHMO) (徐张红等, 2005) (图1)。黎明时分，*CCA1*和*LHY*的转录水平最先达到高峰。CCA1和LHY可通过直接与DET1 (DE-ETIOLATED 1)互作，招募CDD (COP10-DET1-DDB1)复合物发挥转录抑制效应(Lau et al., 2011)。ChIP-Seq数据分析表明，CCA1和LHY可结合在*PRRs*、*ELF3*、*ELF4*、*LUX*和*GI*等生物钟基因启动子区域的EE (AAAATATCT或者AGATATTT)和TCP (GGNCCCAC)元件上，抑制上述基因在清晨表达(Nagel et al., 2015; Kamioka et al., 2016; Adams et al., 2018)。*PRRs*转录起始于*CCA1*/*LHY*之后，按照从早到晚间隔2–3小时的顺序依次表达。*PRR9*/*7*/*5*的转录抑制结构域位于PR (PSEUDO RECEIVER)和CCT (CONSTANS、CONSTANS-LIKE和TOC1)之间的EAR基序。该基序可以招募转录辅阻遏物TPL/TPR (TOPLESS/TOPLESS RELATED PROTEINs)，共同结合在*CCA1*和*LHY*的启动子上抑制其表达(Nakamichi et al., 2010; Wang et al., 2013)。

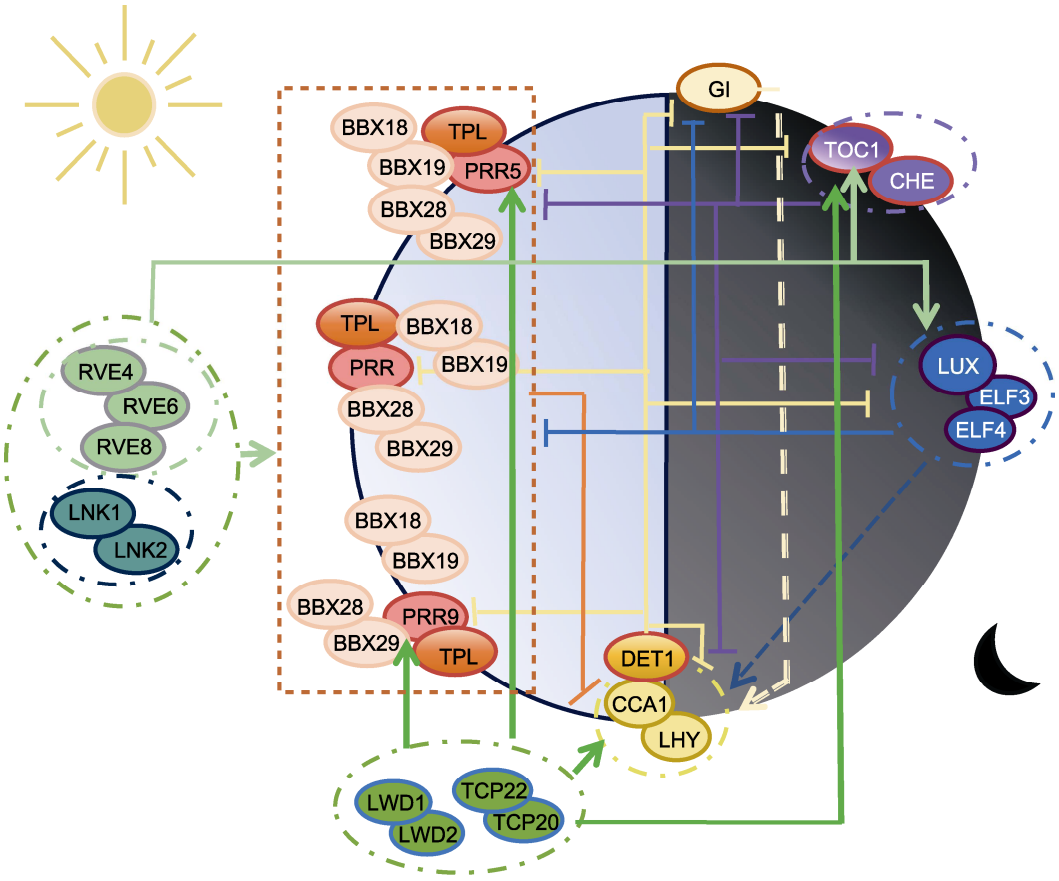


图1 拟南芥生物钟核心振荡器转录-翻译反馈环模式图
箭头表示直接或间接激活作用，钝线表示直接或间接抑制作用。

Figure 1 Transcription-translation feedback loop of *Arabidopsis* circadian core oscillator
Lines with arrows mean direct or indirect activation while blunt lines mean direct or indirect repression.

此外，B-Box第4和第5亚家族成员BBX18 (B-BOX DOMAIN PROTEIN 18)、BBX19及BBX28和BBX29可形成异源二聚体，并次序与PRR9/7/5蛋白在白天形成转录复合体，促进PRR9/7/5对*CCA1*等生物钟核心基因的转录抑制(Yuan et al., 2021a; Yu et al., 2023)。PRR1亦称TOC1 (TIMING OF CAB EXPRESSION 1)，是PRRs家族成员中唯一1个在夜间达到表达高峰的成员。早期研究认为TOC1不具有DNA结合能力，可能通过与TCP型转录因子CHE (CCA1 HIKING EXPEDITION)相互作用，依赖CHE结合到*CCA1*启动子区域的TBS位点(TCP Binding Sites)，从而抑制*CCA1*的表达(Pruneda-Paz et al., 2009)。后续研究证明，TOC1可通过CCT结构域直接结合并抑制*CCA1*、*LHY*、*PRR9*、*PRR7*、*PRR5*、*GI*、*ELF3*、*ELF4*和*LUX*的表达，其转录抑制活性区段位于PR结构域(Gendron et al., 2012; Huang et al., 2012)。*ELF3*、*ELF4*和*LUX*的表达峰值出现在*TOC1*之后，三者具有相似的时间表达模式，常以晚间复合物(evening complex, EC)的形式发挥转录抑制作用。ELF3和ELF4的功能结构域未知，而LUX可以直接结合LBS (LUX Binding Site: GAT($^{A}/_{T}$)CG)位点，抑制*CCA1*、*LHY*、*PRR9*、*PRR7*、*PRR5*以及*GI*的表达(Helfer et al., 2011)。*GI*的表达受到EC、TOC1、CCA1和LHY的抑制，但其可促进*CCA1*和*LHY*的表达(Mizuno et al., 2014; Huang and Nusinow, 2016)。

近年来，研究人员陆续发现一系列转录激活因子参与构成正向转录反馈环。LWD1 (LIGHT-REGULATED WD 1)和LWD2可与TCP (TEOSINTE BRANCHED1-CYCLOIDE-A-PCF)型转录因子TCP20以及TCP22互作并结合到*CCA1*、*PRR9*、*PRR5*和*TOC1*的启动子上激活其表达(Wang et al., 2011; Wu et al., 2016)。中午表达的RVE8 (REVEILLE 8)、RVE6和RVE4可以作为转录激活因子，促进*PRRs*及EC复合体基因的表达(Farinas and Mas, 2011; Rawat et al., 2011; Hsu et al., 2013)。它们也可与LNK1 (LIGHT-INDUCIBLE AND CLOCK-REGULATED 1)和LNK2互作形成复合物，作为转录共激活因子促进*PRR5*、*TOC1*以及*ELF4*的表达(Xie et al., 2014)。反过来，PRRs和EC复合物也可以抑制*LNKs*及*RVEs*的表达(Rugnone et al., 2013; Shalit-Kaneh et al., 2018)。BOA (BROTHER OF LUX ARRHYTHMO)与LUX高度同源但功能不同。研究表明，BOA可直接结合到*CCA1*的启动子上激活其表达，CCA1也可结合到*BOA*的启动子上的EE元件，调控*BOA*的转录水平(Dai et al., 2011)。

综上，生物钟核心组分通过彼此相互连锁调控转录，将转录时相严格限制在特定的时间点，这对于生物钟接收环境信号、维持生物钟节律及调控节律性的生长发育过程不可或缺。

1.1.2 转录后调控

可变剪接体的许多组分已被证实参与生物钟周期调控(图2)。研究发现SKIP (SNW/SKI-INTERACTING PROTEIN)通过与SR45 (SER/ARG-RICH PROTEIN 45)互作，参与调节pre-mRNA 5′和3′端剪接位点的识别和切割。在*skip*突变体中，生物钟周期变长，*PRR7*、*PRR9*、*CCA1*、*LHY*和*TOC1*的可变剪接本发生显著变化(Wang et al., 2012)。snRNP组装因子*GEMIN2* (*GEM NUCLEAR ORGANELLE ASSOCIATED PROTEIN 2*)突变后会导致生物钟周期变短，*TOC1*和*PRR9*的内含子滞留事件增加(Schlaen et al., 2015)。LSM4 (SM-LIKE PROTEIN 4)和LSM5是小分子核糖核蛋白U6 snRNP的组成部分，其突变体生物钟周期延长，*TOC1* (I4R: 第4内含子滞留)和*CCA1* (I2R)的转录本丰度增加(Perez-Santángelo et al., 2014)。*SIC* (*SICKLE*)突变后生物钟周期变长，*CCA1* (I4R)、*LHY* (I1R)、*PRR7* (I4R)以及*ELF3* (I2R)的异常剪接本积累(Marshall et al., 2016)。STIPL1 (SPLICEOSOMAL TIMEKEEPER LOCUS1)参与可变剪接体的解聚过程，其突变后生物钟周期变长，*CCA1*、*LHY*、*PRR9*、*TOC1*和*GI*等生物钟基因积累多种异常剪接本(Jones et al., 2012)。此外，甲基转移酶PRMT5 (PROTEIN ARGININE METHYL TRANSFERASE 5)功能缺失突变体生物钟周期变长，*PRR9* (I3R)转录本丰度增加(Sanchez et al., 2010)。

温度也参与调控生物钟基因的可变剪接(Hernando et al., 2017)。在低温条件下，*LHY*、*TOC1*和*PRR7*转录本中的内含子滞留事件出现频率升高，产生非功能性转录本(James et al., 2018)。*CCA1*基因在不同温度下产生2种不同的转录本，分别为*CCA1α*以及*CCA1β* (图2B)。CCA1β缺少MYB结构域，但是可正常二聚化。高温条件下，*CCA1α*的表达量大幅增

加, *CCA1β*的表达量下降。然而, 低温会诱导*CCA1β*产生, *CCA1α*减少, 同时CCA1β蛋白会竞争性地与全长CCA1α或LHY蛋白互作, 形成不行使功能的复合物, 影响其对下游基因表达的调控, 从而参与冷胁迫的响应过程(Seo et al., 2012)。高温胁迫会促使*CCA1*发生UAS (unproductive alternative splicing)类型的可变剪接, 产生携带提前终止密码子的转录本, 干扰其全长蛋白的正常功能。此外, 在*LUX*、*TIC* (*TIME FOR COFFEE*)以及*LKP2*的转录本中也检测到可变剪接事件, 但尚未证实其生物学意义(Filichkin et al., 2015)。

m^{6}A修饰在调控生物节律的过程中亦发挥重要作用(Fustin et al., 2013), 被誉为新的生物钟调控因子(图2C)。在拟南芥(*Arabidopsis thaliana*)中已鉴定出2种类型的m^{6}A甲基转移酶: 一种是哺乳动物*METTL16*的同源基因*FIO1* (*FIONA1*), 另一种是由MTA (mRNA ADENOSINE METHYLASE)、MTB (METHYLTRANSFERASE B)和FIP37 (FKBP12 INTERACTING PROTEIN 37)等构成的甲基转移酶复合物。研究表明, *FIO1*突变可导致不依赖于环境条件的生物钟周期延长。持续黑暗、红光、蓝光及不同温度条件下, *fio1*突变体均表现为长周期表型(Kim et al., 2008)。进一步研究表明, *fio1*突变体中, *CCA1*和*LHY*部分m^{6}A修饰位点的甲基化水平降低(Xu et al., 2022a)。蓝光受体CRY2 (CRYPTOCHROME 2)通过蓝光诱导的液-液相分离招募m^{6}A编码器MTA复合体(MTA/MTB/FIP37)进入CRY2液相中, 提高MTA蛋白在CRY2液相中的浓度, 进而促进m^{6}A修饰, 调节

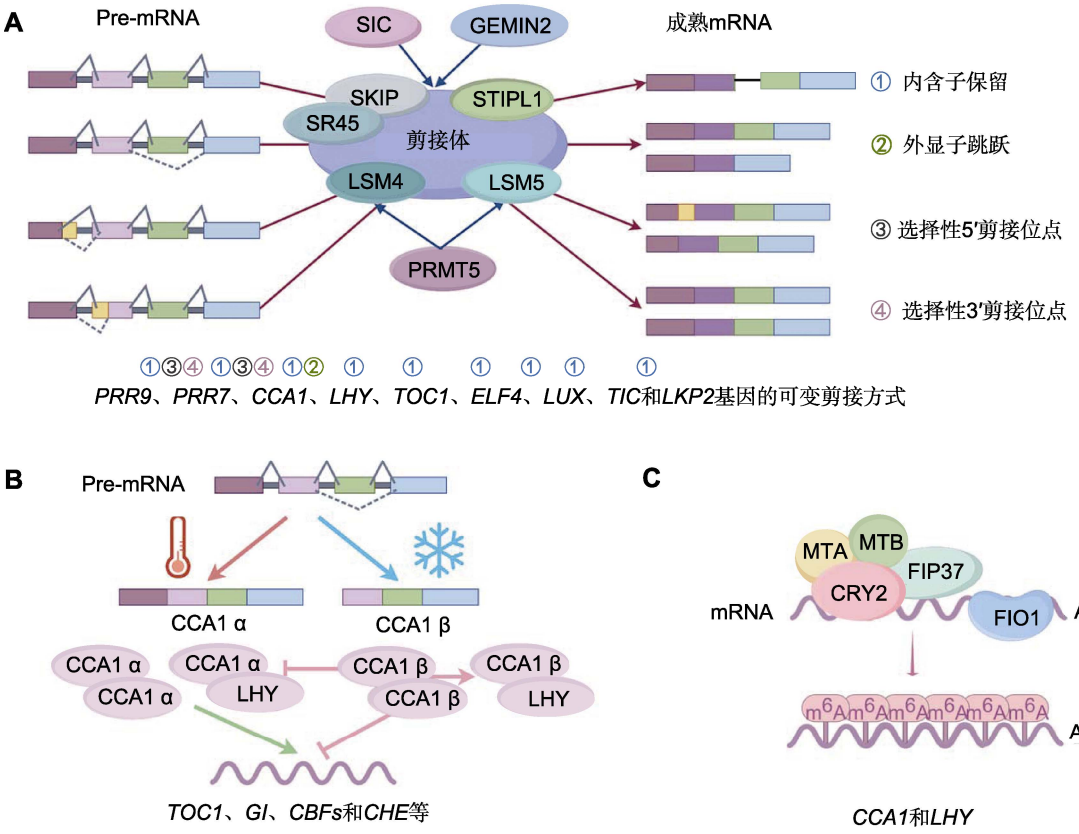


图2 生物钟核心振荡器组分的转录后修饰

(A) 剪接体组分介导拟南芥生物钟基因的可变剪接; **(B)** 温度调控*CCA1*的可变剪接; **(C)** m^{6}A甲基转移酶FIO1和MTA复合体促进*CCA1* mRNA的m^{6}A修饰。

Figure 2 Post-transcriptional modification of the circadian core oscillator components

(A) Components of spliceosome mediate alternative splicing of circadian genes in *Arabidopsis*; **(B)** Temperature regulates the splicing isoforms of *CCA1*; **(C)** m^{6}A methyltransferases FIO1 and the MTA complex promotes m^{6}A installation on *CCA1* mRNA.

m^{6}A修饰的生物钟节律基因*CCA1*的mRNA稳定性, 维持植物的生物钟节律(Wang et al., 2021a)。目前, m^{6}A去甲基化酶是否参与生物钟调控尚未见报道。

1.1.3 翻译后调控

生物钟蛋白存在丰富的翻译后修饰, 这不仅对于蛋白质的成熟、空间构象和理化性质至关重要, 还赋予蛋白质不同的功能, 以动态响应细胞内外的环境变化。

目前, 已报道参与调控生物钟蛋白稳定性的E3泛素连接酶有ZTL (ZEITLUPE)家族蛋白、COP1 (CONSTITUTIVE PHOTOMORPHOGENIC 1)、SDC (SUPPRESSOR OF DRM1 DRM2 CTM3)以及SINAT5。ZTL家族蛋白包括ZTL、FKF1 (FLAVIN-BINDING, KELCH REPEAT, F-BOX 1)和LKP2 (LOV KELCH PROTEIN 2), 它们既是一类蓝光受体又是一种F-box蛋白。三者功能冗余地参与生物钟周期调控, 但ZTL发挥主导作用(Sanchez et al., 2020)。*ztl*突变体无论在持续黑暗还是在持续红/蓝光条件下均具有长周期表型, ZTL过表达则导致生物钟周期变长甚至无节律(Somers et al., 2004)。分子机制上, 研究表明ZTL能直接与TOC1和PRR5互作, 促进二者的泛素化降解, 从而延长生物钟周期(Más et al., 2003; Kiba et al., 2007)。时间上, 研究发现白天蓝光激活ZTL与GI的相互作用, GI一方面招募HSP90 (HEAT SHOCK PROTEIN 90)共同帮助ZTL正确折叠和成熟, 另一方面招募去泛素化酶UBP12 (UBIQUITIN-SPECIFIC PROTEASE 12)和UBP13, 抑制ZTL在白天降解并同时抑制ZTL对TOC1和PRR5的泛素化降解。夜晚来临时, ZTL逐渐从ZTL/GI复合物中解离, 从而促进PRR5和TOC1的降解(Kim et al., 2011; Cha et al., 2017)。此外, PRR3可与ZTL竞争性结合TOC1, 亦在白天抑制ZTL对TOC1的降解(Para et al., 2007)。COP1属于RING-Type E3泛素连接酶, 能与ELF3互作并促进其泛素化, 该过程依赖BBX19 (Wang et al., 2015)。COP1和ELF3均负调控GI蛋白的稳定性, ELF3可能作为“adaptor”介导COP1对GI的泛素化降解(Yu et al., 2008)。SDC是含有F-box结构域的SKP1家族蛋白, 其过表达株系具有长周期的生物钟表型。SDC能与ZTL蛋白互作并促进其降解, 其主要通过ZTL-TOC1级联降解途径来精细调控生物钟周期(Tian et al., 2021)。Song和Carre (2005)研究发现, 在*det1*突变体中, LHY蛋白表达时相提前同时蛋白丰度降低, 这可能是DET1对LHY的转录抑制作用所致。后续实验发现, 与黑腹果蝇(*Drosophila melanogaster*) SINA RING-finger蛋白同源的E3泛素连接酶SINAT, 能与LHY相互作用并促进其泛素化, 而DET1可抑制SINAT5对LHY的泛素化修饰, 但尚无生化实验证据表明SINAT影响LHY蛋白的稳定性(Park et al., 2010)。

目前, 已报道参与调控拟南芥生物钟蛋白磷酸化修饰的激酶包括CK2 (CASEIN KINASE 2)、PPKs (PHOTOREGULATORY PROTEIN KINASEs)和酪氨酸激酶1家族CKLs (CASEIN KINASE 1 LIKEs)。最早鉴定到影响生物钟周期的激酶是CK2, 该激酶属于酪氨酸激酶II家族, 可与CCA1互作并促进其磷酸化。*CCA1-OX*使生物钟周期变长, 振幅降低, 但过表达不能被磷酸化的CCA1几乎均会影响生物钟周期和振幅, 说明CCA1磷酸化为其发挥作用所必须(Daniel et al., 2004)。PRRs在一天当中具有明显的动态磷酸化修饰(Fujiwara et al., 2008)。Uehara等(2019)研究发现, 以CKL4为代表的CKLs家族成员能与TOC1和PRR5互作, 并促进二者的磷酸化和降解。使用磷酸化酶抑制剂PHA767491处理, 会导致TOC1和PRR5蛋白累积并伴随二者靶基因的表达量下降。PPKs (PPK1、PPK2、PPK3和PPK4)属于酪氨酸激酶I家族, 为红光诱导的PIF3 (PHYTOCHROME INTERACTING FACTOR 3)磷酸化和降解所必须(Ni et al., 2017), 同时能促进蓝光受体CRY2磷酸化。而CRY2可竞争性抑制PPKs对PRR9的磷酸化修饰, 影响其转录活性和DNA结合能力, 从而使生物钟感知光强信息变化(He et al., 2022)。PPKs亦称MLKs (MUT9-LIKE KINASEs), 质谱数据表明, ELF3能够与MLKs互作(Huang et al., 2016), 但MLKs是否参与磷酸化修饰ELF3尚不明确。此外, O-岩藻糖基化修饰转移酶SPY (SPINDLY)可与PRR5互作并糖基化修饰PRR5, 通过促进其降解调控生物钟周期(Wang et al., 2020a)。

综上, 目前研究发现, 泛素化、磷酸化和糖基化等翻译后修饰参与调控拟南芥生物钟核心蛋白在一天当中的动态变化, 并为维持蛋白的稳定性或DNA结合能力所必须。

1.1.4 表观遗传调控

多数生物钟基因, 如*CCA1*、*LHY*、*PRR9*、*PRR7*、

TOC1、*GI*和*LUX*的启动子区域均存在乙酰化和甲基化修饰，并且一天当中的动态变化与其表达水平密切相关(图3)。

细胞内组蛋白的乙酰化和去乙酰化过程处于动态平衡，由乙酰转移酶HAT (HISTONE ACETYLTRANSFERASE)和去乙酰化酶HDAC (HISTONE DEACETYLASE)共同调控。*TOC1*启动子的乙酰化水平在清晨较低，夜晚较高，这与其表达水平一致。分子机制上，CCA1/LHY与组蛋白修饰复合物LDL1 (LYSINE-SPECIFIC DEMETHYLASE 1)、LDL2以及HDA6 (HISTONE DEACETYLASE 6)相互作用，结合在*TOC1*的启动子上并降低其H3K4me3和H3Ac的修饰水平，从而抑制*TOC1*在白天表达(Hemmes et al., 2012; Hung et al., 2018); RVE8/LCL5 (LHY-CCA1-LIKE5)则与CCA1/LHY具拮抗作用，其通过与*TOC1*的启动子结合促进H3乙酰化，从而激活*TOC1*的表达(Farinas and Mas, 2011)。需要说明的是，TOC1也可以结合LDL1/LDL2/HDA6复合物，降低*CCA1*/*LHY*启动子区域的H3K4me3和H3Ac修饰水平，从而抑制*CCA1*/*LHY*的转录(Hung et al., 2019)。PRR5/7/9通过其保守的EAR基序招募TPLs及HDA6，通过降低*CCA1*和*LHY*启动子区域的乙酰化水平，抑制二者的表达，从而维持生物钟周期(Wang et al., 2013)。CCA1在清晨激活*HAF2* (*HISTONE ACETYLTRANSFERASE OF THE TAFII250 FAMILY 2*)的表达，促进其在中午达到表达高峰。HAF2蛋白结合*PRR5*和*LUX*基因的启动子，促进H3ac沉积，从而激活二者的表达(Lee and Seo, 2018)。

组蛋白甲基化由HMT (HISTONEMETHYL TRANSFERASE)催化赖氨酸和精氨酸残基完成，组蛋白去甲基化酶主要包括LSD1 (LYSINE SPECIFIC DEMETHYLASE 1)和JmjC (JUMONJI C)家族。研究表明，组蛋白甲基转移酶SDG2/ATXR3 (SET DOMAIN GROUP 2/ARABIDOPSIS TRITHORAX RELATED 3)直接或间接调控*CCA1*、*LHY*、*PRR9*、*PRR7*、*TOC1*、*GI*和*LUX*基因启动子区域的H3K27me3、H3K27me2和H3K9me3水平，从而调控其表达(Malapeira et al., 2012)。组蛋白去甲基化酶基因*JMJD5* (*JUMONJI DOMAIN CONTAINING 5*)以及*JMD30*参与生物钟周期调控，二者的突变体均导致生物钟周期加快。CCA1可以直接结合到*JMJD5*和*JMD30*的启动子上抑制其表达。而JMJD5和JMD30可与TOC1相互作用，共同抑制*CCA1*以及*LHY*的表达(Jones et al., 2010; Lu et al., 2011)。DNA甲基化修饰也为维持生物钟节律所必须，DNA甲基转移酶突变体及利用DNA甲基化抑制剂处理的野生型拟南芥生物钟周期均在持续光照下显著延长(Tian et al., 2021)。

此外，蛋白激酶MLK3和MLK4可通过与CCA1互作结合到*GI*的启动子上，介导H2AS95磷酸化。在*mlk4*突变体中，*GI*启动子区域的H2AS95磷酸化减少并伴随H2A.Z和H4乙酰化水平降低，抑制*GI*的表达，从而导致晚花发育表型(Su et al., 2017)。E3泛素连接酶HUB1 (HISTONE MONOUBIQUITINATION 1)不参与蛋白质降解，而是介导与植物转录激活有关的组蛋白H2B单泛素化修饰及H3甲基化修饰。转录组数据分析表明，HUB1参与调控*GI*、*PRR5*、*ELF4*、*CCA1*和*LHY*的表达，在*hub1*突变体中*ELF4*、*CCA1*和*TOC1*基因编码区的H2B单泛素化修饰水平降低(Himanen et al., 2012)。

综上，组蛋白在相关酶的作用下可发生乙酰化、甲基化、磷酸化和泛素化等不同的化学修饰，进一步影响染色质结构或招募不同的互作因子来调控生物钟基因的转录活性。

2 生物钟对光信号的感知与响应

光信号并非是一成不变的环境信号，基于季节、纬度和时间等因素的变化，光周期、光质和光强信息也会产生相应的改变。光受体将光信息转化为化学信号，是植物接受和传递光信号变化的第1步。拟南芥中的光感受器至少包含5个家族，分别为光敏色素PHYs (PHYTOCHROMEs)、隐花色素CRYs (CRYTOCHROMEs)、ZTL家族(ZTL、FKF1和LKP2)、向光素PHOTs (PHOTOTROPINs)以及UVR8 (UV RESISTANCE LOCUS 8)。近年来，对拟南芥光受体和生物钟分子互作网络的研究表明，光受体与生物钟组分间存在复杂的调控网络，且部分光受体与生物钟组分间的互作状态是植物感知光周期、昼夜转换及光强信息变化的重要方式，对于调控下游节律性生长发育过程也至关重要。

2.1 光周期信号与生物钟

2.1.1 光受体与生物钟互作调控光周期依赖的开花时间

植物对于白天和黑夜相对长度的反应称为光周期现

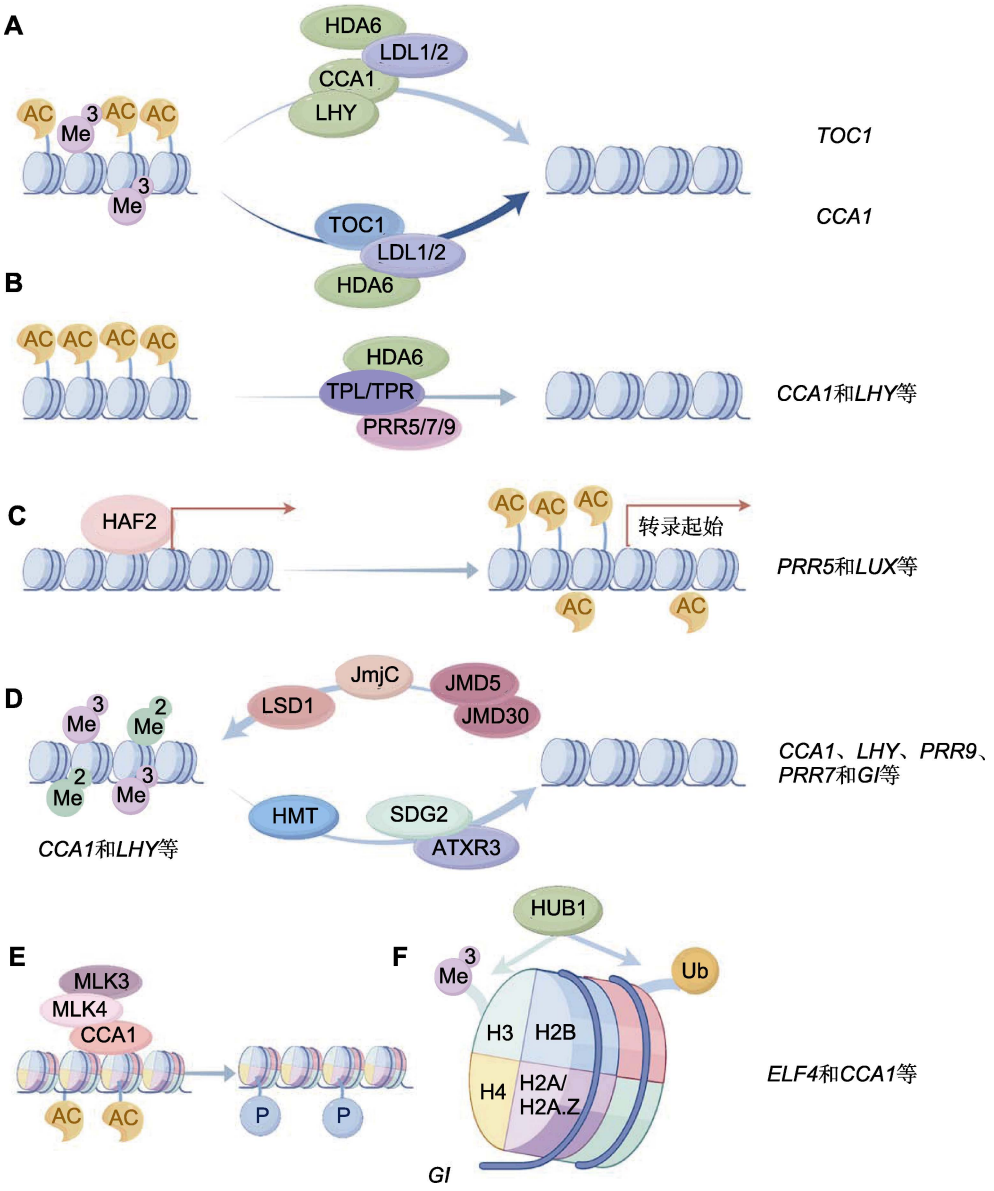


图3 生物钟核心振荡器组分的染色质修饰水平调控模式图

(A) CCA1/LHY或TOC1与LDL1/2-HDA6复合物结合抑制彼此的乙酰化(AC)和三甲基化(Me^3)修饰; **(B)** PRR5/7/9招募TPL/TPR和HDA6去除靶基因启动子区域的乙酰化; **(C)** HAF2结合靶基因的启动子区域, 促进H3AC沉积, 激活基因的表达; **(D)** 组蛋白去甲基化酶LSD1、JmjC、JMJD5/JMD30及甲基转移酶HMT、SDG2/ATXR3直接或间接调控生物钟基因启动子区域的甲基化水平; **(E)** CCA1介导MLK3和MLK4调控*GI*启动子区域的H2AS95磷酸化(P)和H4乙酰化; **(F)** HUB1参与调控靶基因启动子区域的H2B单泛素化(Ub)和H3甲基化修饰。

Figure 3 A diagram showing chromatin modifications regulation of the circadian core oscillators

(A) CCA1/LHY or TOC1 interacts with LDL1/2-HDA6 complex to inhibit the acetylation (AC) and trimethylation (Me^3) modifications of each other; **(B)** PRR5/7/9 recruits TPL/TPR and HDA6 to remove the acetylation modification of target genes; **(C)** The histone acetyltransferase HAF2 binds to the promoter region of the target genes to promote the deposition of H3AC and activate gene expression; **(D)** The histone demethylase LSD1, JmjC, JMJD5/JMD30, and the methyltransferase HMT, SDG2/ATXR3 directly or indirectly regulate the methylation level of the promoter region of clock genes; **(E)** CCA1 mediates the regulation of MLK3 and MLK4 on the phosphorylation (P) of H2AS95 and the acetylation of H4 in the *GI* promoter region; **(F)** HUB1 is involved in regulating the monoubiquitination (Ub) of H2B and the methylation modification of H3 in the promoter regions of target genes.

象，光周期影响植物生长发育的很多方面，其中最显著的是严格限制植物的开花时间。多数植物必须在生长发育的某一阶段经历足够长的适宜光周期才能起始开花，否则将保持在营养生长期。目前，对拟南芥的研究表明，光受体与生物钟组分间的协调互作在介导光周期依赖的开花调控中发挥重要作用。

成花素基因*FT*是诱导植物开花的关键因子。长日照下，*FT*的表达水平较高，促进拟南芥开花。与之相反，短日照下，*FT*的表达水平极低，抑制开花。日长依赖的*FT*表达模式主要由开花途径的核心因子CO (CONSTANS)调控。CO主要作为转录激活因子，激活成花素基因*FT*的表达，从而促进开花(Song et al., 2015)。在转录水平上，CDF1 (CYCLING DOF FACTOR 1)是第1个被发现调控*CO*的转录因子，属于CDFs家族(CDF1–CDF5)。CDFs在早晨结合在*CO*的启动子上并募集转录共抑制因子TPL来抑制*CO*的表达。在*cdf1*/*cdf2*/*cdf3*/*cdf5*四突变体中，无论长短日照，*CO*的转录水平在清晨均显著上升，暗示对*CDFs*转录水平的调控也是拟南芥感知光周期信息变化的重要一环(Fornara et al., 2009; Goralogia et al., 2017)。

后续研究表明，CO及CDFs家族成员的转录和蛋白稳定性受到生物钟与光受体协同调控(图4)。在转录水平上，CCA1/LHY在清晨诱导*CDFs*的转录，PRR9/7/5在下午起相反作用，抑制其转录，从而将*CDFs*的表达时相限制在清晨(Nakamichi et al., 2007, 2012; Song et al., 2015)。在转录后水平上，CDFs蛋白的稳

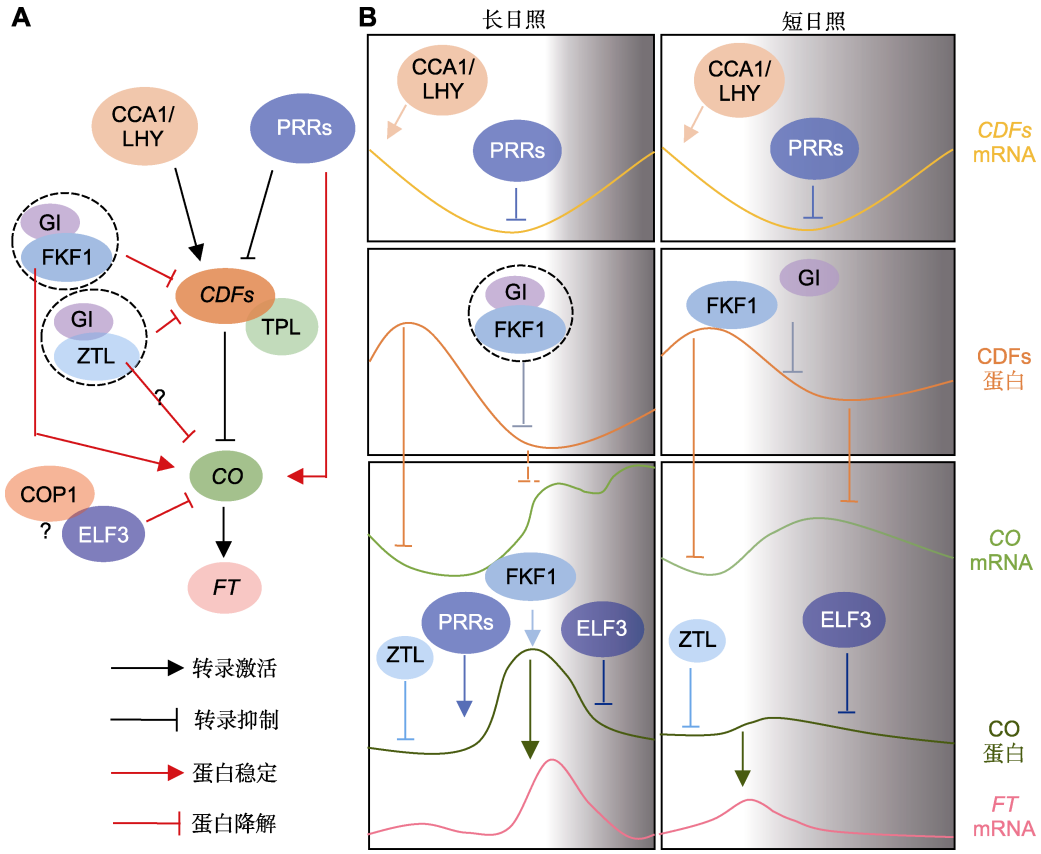


图4 拟南芥生物钟调控光周期依赖的开花模式图

(A) 生物钟组分通过调控*CO*-*FT*模块介导开花的简易模型(黑线代表转录调控，红线代表蛋白调控)；**(B)** 不同光周期下生物钟组分差异调控*CO*-*FT*表达的时相示意图。?: 未知

Figure 4 The diagrams showing photoperiodic flowering regulated by the circadian clock in *Arabidopsis*

(A) A simplified model of flowering mediated by the clock components through regulation of *CO*-*FT* modules (the black lines represent transcriptional regulation, the red Lines represent protein regulation); **(B)** Schematic diagrams of how the clock differentially regulate the expression of *CO*-*FT* under different photoperiods. ?: Unknown

定性受FKF1和GI严格调控。长日照下, FKF1和GI的表达时相耦合, 均在下午达到峰值。FKF1以蓝光依赖的方式与生物钟分子伴侣蛋白GI互作, 形成足够的FKF1/GI复合物并促进CDFs蛋白降解, 释放对*CO*的转录抑制, 从而促进开花。短日照下, 由于FKF1和GI的表达时相存在相位差, 使得FKF1/GI复合物的形成在白天极大减少, CDFs的降解微乎其微, *CO*始终处于低水平状态, 从而延迟开花(Sawa et al., 2007; Fornara et al., 2009)。除FKF1外, 其家族成员ZTL及LKP2也能与GI互作, 参与降解CDF2, 从而解除对*CO*的转录抑制(Kim et al., 2007)。目前, 普遍认为FKF1/GI-CDFs-*CO*信号通路是光受体与生物钟协同互作感知光周期变化, 从而调控开花时间的经典方式。

FKF1不仅可通过与GI形成复合物调控CDFs蛋白的稳定性来达到间接调控CO的表达水平, 也可直接与CO蛋白互作来稳定CO蛋白(Song et al., 2012)。与之相反, 早晨***ztl***突变体中的CO能稳定存在, 可能由于ZTL直接与CO互作并降解CO蛋白(Hwang et al., 2019)。生物钟组分在转录后水平上对CO蛋白稳定性的调节同样起着重要作用。研究表明, PRRs家族蛋白可在一天中从早到晚分别与CO蛋白互作并稳定CO, 促进长日照下CO的积累与开花提前(Hayama et al., 2017)。在远红光(FR)辐照度相对较低(R/FR> 2.0)的实验室条件下, ELF3能够与CO互作并促进CO蛋白降解(Song et al., 2018), 原因可能是其通过与COP1互作来促进这一过程。在*gi*/*elf3*双突变体中, 生物钟介导的光周期响应的开花时间调控途径丧失作用, 表明GI和ELF3协同将光周期信号传递给中央振荡器(Anwer et al., 2020)。

2.1.2 光周期调控生物钟基因表达相位介导光形态建成

光周期不仅影响植物的开花时间, 也调控下胚轴的节律性生长。短日照条件下, 下胚轴加速伸长, 并在黎明时伸长速率达到峰值。PIFs是调控下胚轴生长的关键因子, 其中PIF4和PIF5起主效作用(Leivar and Monte, 2014)。下胚轴节律性生长由多方面因素介导: 首先, *PIF4*/*5*的表达受到生物钟调控, 一般在夜间达到峰值, 长夜周期会使*PIF4*/*5*积累量更高(Nozue et al., 2007; Nusinow et al., 2011); 其次, PIFs蛋白在白天被激活的光敏色素蛋白降解, 在晚间积累(Soy et al., 2012, 2016)。此外, PIFs的活性受到多个生物钟组分的拮抗作用, 如PRRs、GI和EC复合物。最后, PIFs直接诱导下游生长促进因子的表达, 促进下胚轴生长(图5)。

早间组分*cca1*/*lhy*双突变体在不同光周期下均为短下胚轴表型, 这可能与不同的光周期下*PIF4*/*5*的表达相位改变有关(Niwa et al., 2009)。持续红光下, CCA1/LHY与SHB1 (SHORT HYPOCOTYL UNDER BLUE 1)互作, 增强对*PIF4*的转录激活, 从而在早间调控下胚轴生长(Sun et al., 2019)。生物钟核心组分PRRs功能缺失突变体在不同光周期下均呈现长下胚轴表型。PRRs通过结合*PIF4*/*5*启动子上的G-box元件抑制其表达。同时PRRs家族成员的蛋白水平会随着光周期在相位和丰度上发生改变(Li et al., 2020), 表明PRRs可以感知并传递日长信息。因此, PRRs是调控光周期依赖的下胚轴生长的关键因子。拟南芥在短日照下黎明前显著增长的下胚轴表型可能是由于短日照下PRR9/7/5能够从早晨到黄昏与PIFs蛋白互作并抑制其转录活性, 从而抑制下游基因*CDF5*的表达; 夜间PRR9/7/5降解, PIFs蛋白结合*CDF5*的启动子并诱导其表达, 使之在黎明前特异性积累, 诱导细胞伸长(Martín et al., 2018)。此外, TOC1也可通过在细胞核中与PIF3互作, 抑制其转录激活活性, 调节生长相关基因的表达(Soy et al., 2016)。TOC1一般在黄昏后达到表达高峰, 并在夜间至黎明逐渐降解, 因此其对*PIF3*/*4*的转录水平及转录活性的抑制主要集中在晚间。而去磷酸化的TOC1会更早出现, 更快降解, 与NF-YC (NUCLEAR FACTOR Y subunits C)的互作减少导致染色质驻留减少, 同时TOC1的活性降低也减弱了其与PIF3和HDA15的互作, 使下胚轴更早且更快生长(Yan et al., 2021)。

EC复合体通过结合*PIF4*/*5*的启动子, 在夜间早期抑制*PIF4*/*5*的转录(Nusinow et al., 2011)。ELF3蛋白也可与PIF4蛋白互作抑制其DNA结合活性(Nieto et al., 2015)。BBX19促进ELF3被COP1降解, ELF3的消耗导致EC复合物形成减少, 使得*PIF4*/*5*在傍晚早期表达水平更高, 从而促进下胚轴伸长(Wang et al., 2015)。*gi*突变体在不同光周期下均呈现长下胚轴表型, 大数据分析发现GI和PIFs在一天结束的时间点协同调控许多参与光形态建成基因的表达(Kim et al., 2012)。GI不仅在转录水平上影响*PIFs*的表达, 还

调控PIFs蛋白质的稳定性和转录活性。随着GI在深夜的降解，PIFs蛋白逐渐富集，在黎明前后促进下胚轴生长(Nohales et al., 2019)。此外，GI和ELF4可影响*PIF4*的表达，控制不同光周期下的下胚轴生长(Kim et al., 2012)，但具体机制尚不清楚。

2.2 光质信号与生物钟

不同光质信号既可通过光受体与生物钟组分直接互作，也可通过光受体-光信号枢纽因子-生物钟组分的级联信号通路向生物钟输入。

PHYA和PHYB分别为介导远红光和红光信号重置生物钟所必须(Yanovsky et al., 2001)。CHIP数据表明，PHYB偏好结合G-box基序，可直接结合*GI*和*PRR9*d的启动子，可能是一种转录抑制因子(Jung et al., 2016)。此外，PHYB可能与ELF3、LUX、CCA1、LHY、TOC1和GI相互作用(Yeom et al., 2014)，但其与生物钟组分互作的调控出口仍不清楚。PHYA虽然缺乏已知的DNA结合结构域，但是有模型提出PHYA可作为非典型转录因子与靶基因结合(Chen et al., 2014)。Li等(2011)研究发现，远红光信号通路中的2个转录因子FAR1 (FAR-RED IMPAIRED RESPONSE 1)和FHY3 (FAR-RED ELONGATED HYPOCOTYLS 3)为维持*ELF4*节律和振幅所必需。此外，二者还可通过直接结合*CCA1*的启动子激活其表达，而PIF5和TOC1可通过与FHY3和FAR1互作抑制二者对*CCA1*表达的激活(Liu et al., 2020)。反之，CCA1

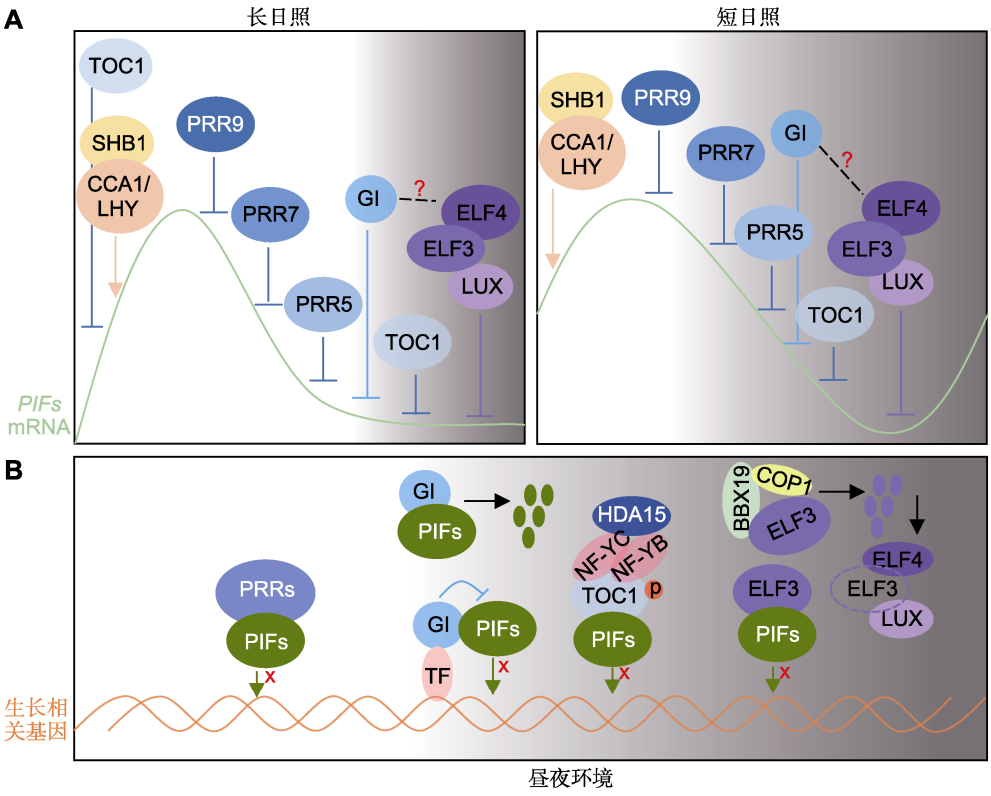


图5 拟南芥生物钟调控光周期依赖的下胚轴生长模式图

(A) 不同光周期下生物钟差异调控*PIFs*转录水平的模式图(箭头代表转录激活，钝线代表转录抑制)；**(B)** 昼夜环境下，生物钟调控PIFs蛋白水平和转录活性示意图。“x”代表干扰转录激活活性，“p”代表磷酸化，多个同色小圆代表该蛋白降解。?: 未知

Figure 5 The diagrams showing photoperiodic hypocotyl growth regulated by the circadian clock in *Arabidopsis*

(A) Schematic models of the clock differentially regulate the expression of *PIFs* transcriptionally under different photoperiods (the arrows represent transcriptional activation, and the lines represent transcriptional inhibition); **(B)** A drawing of the clock regulation of PIFs protein levels and transcriptional activity in diurnal condition. “x” represent interference of transcriptional activation ability, “p” represent phosphorylation, and multiple small circles of the same color represent the degradation of the protein. ?: Unknown

和TOC1可通过直接与FHY3互作，抑制其对下游靶基因*FHY1*的转录活性，从而反馈调控PHYA介导的远红光信号通路(Liu et al., 2022)。与此类似, Wang等(2022a)研究发现，生物钟调节子TIC可反向调节光受体，其招募共转录抑制因子TPL，通过未知转录因子结合在*PHYA*的启动子上负调控其表达，且TIC能直接与PHYA互作，影响其稳定性和核小体的形成。

避荫反应是一种由光质改变引起的自然现象，主要由于自然环境下叶片遮挡导致局部光环境中FR比例上升(R/FR降低)，造成PHYB、PHYD和PHYE光感受器失活，从而稳定PIF家族并促进下胚轴或茎的伸长。生物钟也参与遮阴环境下的光形态建成。在高遮阴环境下(R:FR<0.1)，黄昏诱导*TOC1*的转录水平高低依赖于PHYA，而RVE8可能是PHYA转录调控*TOC1*所必需的转录因子，但具体机制并不清楚(Fraser et al., 2021)。Fraser等(2021)研究发现，*PRR7*、*ELF3*和*ELF4*也以PHYA依赖的形式在高遮阴环境下被诱导表达，可能通过结合*PIF*的启动子抑制其转录活性，调控下游生长因子，进而参与调控下胚轴生长。PIF家族对遮阴环境中的下胚轴调控存在功能冗余，但PIF7可能起主效作用(Jiang et al., 2019)。遮阴条件下，PRRs也通过与PIFs家族成员互作干扰其对下游基因的转录活性，负调控下胚轴生长(Martín et al., 2018)。ELF3在遮阴环境介导的下胚轴生长中发挥更关键的作用，可与PIF7互作并干扰其结合DNA的能力，以抑制遮阴诱导的*IAA29* (*INDOLE ACETIC ACID-INDUCIBLE 29*)以及*PIL1* (*PHYTOCHROME INTERACTING FACTOR 3-LIKE 1*)表达，从而调控下胚轴生长(Jiang et al., 2019)。

He等(2022)研究表明，蓝光信号输入生物钟主要依赖蓝光受体CRYs和ZTL。CRY2 (而非CRY1)特异地与PRR9互作，介导蓝光信号输入，而CRY1的作用机制并不清楚。ZTL主要通过GI/ZTL-TOC1/PRR5介导蓝光信号调控生物钟周期(Kim et al., 2013; Cha et al., 2017)。此外, ZTL还参与泛素化降解CHE (Lee et al., 2018)。进一步研究发现，光信号转导中起分子枢纽作用的转录因子HY5及其同系物，也是将蓝光信息输入生物钟的关键整合者。蓝光更易诱导*HY5* mRNA和蛋白积累，从而调控其与生物钟基因启动子的结合, HY5直接调控*PRR5*、*LUX*和*BOA*的表达，并推测其在转录后水平调控*CCA1* (Hajdu et al., 2018)。

UV-B光如何输入生物钟目前仍然未知，但有研究发现UV-B光脉冲依赖UVR8和COP1诱导*CCA1*/*LHY*等核心时钟基因的转录。该过程的转录诱导水平又取决于施加脉冲的时间，表明这种反应可能由生物钟门控，由此建立UV-B光感知生物钟间反馈调节回路(Fehér et al., 2011)。

2.3 光强信号与生物钟

光照强弱对于昼夜振荡的速率也至关重要。植物在不同光照强度下的生物钟周期曲线称为光频率响应曲线(fluence response curve, FRC)。光照强弱驯化生物钟周期的速率遵循Aschoff定律：对于有昼夜节律的昼行性动物和植物而言，在恒定的光照下，光强与生物钟周期的长短呈负相关，即光强越强周期越短。对于夜行性动物而言，光强则与生物钟周期的长度呈正相关(Aschoff, 1979)。然而，截至目前，在时间生物学领域关于Aschoff定律的分子机制仍不甚明晰。

20世纪初，有研究初步分析了部分光受体在不同光质下的FRC曲线，结果表明PHYA、PHYB、CRY1和CRY2均参与条件依赖的光强信号驯化生物钟的过程(Somers et al., 1998; Devlin and Kay, 2000)。Fehér等(2011)研究表明UVR8也介导UV-B光驯化生物钟。向光素并不参与驯化生物钟的过程(Litthauer et al., 2015)。然而，对于上述光受体具体介导何种光质、多大区间内的光强以及调控生物钟周期快慢的分子机制仍待进一步探索。

Farré等(2005)发现*prr9-1*突变体在持续红光下的FRC曲线遵循Aschoff定律，而在持续蓝光下的FRC曲线有悖于Aschoff定律，这说明PRR9的功能与光强密切相关。He等(2022)的研究进一步证明在2.5–10 μmol·m^{-2}·s^{-1}弱蓝光区间，随着光强的增强，*prr9*突变体的生物钟周期不降反升，说明PRR9为弱蓝光下维持Aschoff定律所必需的生物钟因子。弱蓝光下，CRY2能够与PRR9互作并干扰PRR9-PPKs及PRR9-TPL复合物的形成，减弱PRR9的磷酸化修饰、DNA结合能力及对靶基因的转录抑制作用，从而造成生物钟周期延长；强蓝光下，由于CRY2蛋白降解，释放了对PRR9的双重功能抑制，从而导致生物钟节律较快(He et al., 2022)。由此说明，蓝光受体CRY2能够与生物钟组分PRR9互作，使拟南芥感知光强信息从而维持适宜的生物钟快慢周期。

3 生物钟对温度信号的感知与响应

与光信号相同，温度也是重要的授时因子，具有昼夜性和季节性变化规律。一方面，温度可驯化生物钟，即在持续光照或黑暗下，给予植物一定的温度循环，内源生物钟能维持近似24小时的昼夜节律；给予瞬时的温度刺激可引起生物钟相位或振幅变化。另一方面，生物钟具有温度补偿性(temperature compensation, TC)，即生物钟相对稳定，具有很强的抗干扰能力，面对波动的环境温度(生理温度范围内)依然能够维持周期近似24小时恒定(Buhr et al., 2010; Avello et al., 2019)。温度驯导和补偿分别是生物钟门控温度信号输入和输出的两个方面，二者相互依赖，密不可分。

3.1 温度驯导

暴露于自然环境中的植物，需要应对不断变化的环境温度，因而植物对温度信号的感知以及早期信号向生物钟系统传递的过程，对于生物钟调节下游基因的表达进而调节生长发育状态至关重要。对于拟南芥生物钟系统而言，温度可通过不同层面，直接或间接作用于不同的生物钟系统组分，启动温度信号应答。

(1) 温度诱导生物钟基因染色质结构重塑，进而影响转录效率。研究表明H2A.Z参与温度响应以及基因表达调控。当温度升高时，H2A.Z从温度响应基因的核小体上移除，从而促进温度响应基因的转录(Kumar and Wigge, 2010; Cortijo et al., 2017; Xue et al., 2021)。因此，从DNA或染色体结构层面而言，H2A.Z有可能是温度受体。生物钟EC复合体成员ELF3可直接与SEF互作，招募SWR1 (SWI2/SNF2-RELATED)染色质重塑复合物，调节EC靶基因结合位点区域H2A.Z的沉积。EC靶基因*PRR7*和*PRR9*启动子结合位点上的H2A.Z沉积在白天少夜间多，与二者的表达节律相吻合(Tong et al., 2020)，说明温度信号可以通过直接调节生物钟基因启动子区域的H2A.Z置入和移除影响转录效率。

(2) 温度可直接或间接调控部分生物钟基因的转录和可变剪接。热休克因子在高温胁迫应答调控中发挥非常重要的作用，高温激活热激转录因子的表达进而激活转录水平的级联调控(Yoshida et al., 2011; Quint et al., 2016)。Kolmos等(2014)研究表明，热休克蛋白HsfB2b (heat shock transcription factor B2b)可直接结合*PRR7*的启动子并抑制其表达，该过程可能参与介导高温信号到生物钟核心振荡器的输入及温度对生物钟系统的重置。CBFs (C-REPEAT/DRE BINDING FACTOR s)是植物应答低温信号的核心转录因子，低温可诱导RVE4和RVE8从细胞质进入细胞核激活*CBFs*的表达，同时促进其转录抑制因子CCA1/LHY的降解，从2个层面促进CBFs的累积，进而提高冷胁迫应答能力(Dong et al., 2011; Kidokoro et al., 2021)。此外，低温信号可促进生物钟调控基因*COR27* (*COLD-REGULATED GENE27*)以及*COR28*的表达，*cor27*/*cor28*双突变体的抗冷胁迫能力提高(Li et al., 2016)。另外，温度变化还影响大部分生物钟基因的可变剪接过程。

(3) 温度可影响光敏色素互作因子*PIF7* mRNA的二级结构变化，从而影响翻译效率。*PIF7* mRNA翻译起始位置存在1个发卡结构，高温可部分解开该结构，促使其mRNA的翻译效率提高。PIF7响应高温信号，进一步促进热响应基因的表达，从而促进下胚轴伸长(Chung et al., 2020)。生物钟组分ELF3能与PIF7互作并抑制其DNA结合能力，从而参与调控避荫反应(Jiang et al., 2019)。其它生物钟基因的RNA二级结构是否受温度直接调控尚无系统研究。

(4) 温度可改变蛋白的活性或构象。随着环境温度的升高，*PHYB*的转录和蛋白水平无显著变化，但是PHYB的构象会从Pfr向Pr转变，细胞核内Pfr-Pfr二聚体和光小体的丰度显著降低，从而促进热形态建成相关基因的表达(Jung et al., 2016; Legris et al., 2016)。后续研究发现，活化的PHYB可通过温控液-液相分离感知温度信号。PHYB的NTE区段含有IDR序列，是其响应温度的特异序列，而C端通过寡聚作用促进光小体的形成。当PHYB处于Pr状态时，NTE被包埋于N端感光区，抑制C端的寡聚能力，从而抑制PHYB光小体的形成。PHYB可通过LLPS动态可逆地形成和解离光小体，调整其信号通路的转导效率，快速响应环境温度变化。需要说明的是，PHYB能直接与ELF3互作并正调控其稳定性(Liu et al., 2001)，但二者介导温度信号向下游传递的分子遗传关系尚不清楚。

(5) 温度可诱导ELF3发生液相分离。由ELF3、ELF4和LUX组成的晚间复合物是环境温度信号输入生物钟的关键整合因子(Huang and Nusinow, 2016;

Ezer et al., 2017)。Jung等(2020)研究表明, 生物钟的核心组分ELF3也是一种温度受体, 其polyQ (Polyglutamine)区域含有类朊病毒结构, 介导高温下ELF3的相分离过程, 从而改变其DNA结合活性和转录抑制作用。温度也能调控ELF4从茎向根的移动速度, 低温有助于其从茎向根部移动, 造成根部生物钟变慢, 高温则会抑制ELF4的移动从而使根部生物钟变快(Chen et al., 2020)。温度还参与调控EC的DNA结合亲和力及其对靶基因的转录调控活性, 低温下EC结合DNA的能力较强, 随着温度的升高, 其DNA结合能力降低。体外实验表明, LUX自身具有较强的DNA结合能力, 而LUX-ELF3的DNA结合能力较差, 过表达ELF4可增强EC的DNA结合活性(Silva et al., 2020)。

(6) 温度可直接调控ZTL泛素连接酶的稳定性。高温可促进ZTL蛋白累积, 并进一步招募分子伴侣蛋白HSP90, 促进细胞内错误折叠蛋白的再折叠并清除胞内毒性聚合物, 从而稳定高温下的生物钟系统, 提高植物应对热胁迫的能力(Gil et al., 2017)。

尽管上述研究表明, 温度可从不同基因及不同层面引起生物钟基因的表达变化, 但并不能回答在恒定的光照或黑暗下, 温度循环如何驯导生物钟。Salomé等(2010)研究表明, PRR7/PRR9参与条件依赖的温度循环驯导生物钟过程, 但具体的分子机制并不清楚。

3.2 温度补偿

生物钟如何维持温度补偿的周期特性一直是亟待解决的重要问题。为鉴定拟南芥中参与温度补偿的生物钟组分, 科研人员观察和筛选了12°C、22°C和27°C下不同生态型间的叶片运动差异, 定位到9个影响温度补偿的数量性状位点, 包括*FLC*、*GI*以及*ZTL*等(Edwards et al., 2005)。Gould等(2006)后续研究表明, GI为维持高温和低温下的生物节律所必须。*FLC*功能缺失后特异性在高温下表现出长周期表型, 而该过程可能通过改变*LUX*基因表达峰值的水平参与温度补偿(Edwards et al., 2006)。*CCA1*和*LHY*突变也部分丧失温度补偿。与其它突变体不同的是, *prr79*双突变体的生物钟周期在高温下出现过补偿, *amiR-CCA1-LHY*/*prr79*的周期表型则与各个温度下*amiR-CCA1-LHY*短周期的表型一致, 说明*CCA1*/*LHY*与*PRR7*/*9*调控生物钟温度补偿处于同一遗传学通路, 但具体分子机制未知(Salomé et al., 2010), 这也说明PRR7/9是介导温度信号驯导生物钟和维持温度信号稳定输出的关键整合因子。此外, RVE8对生物钟周期的调控也受温度影响, 在22°C和27°C下, *rve8*突变体的生物钟周期相比野生型变长, 而在17°C、22°C和27°C下*RVE8-OE*的生物钟周期均显著变短, 仅在12°C下表现出类似野生型的周期表型(Rawat et al., 2011)。JMJD5也参与温度补偿过程, 随着温度的升高, *JMJD5*的mRNA及蛋白水平上升(Gan et al., 2014); *JMJD5*功能缺失后在高温下表现出更为显著的短周期表型, 该过程可能通过改变高温下生物钟基因*PRR7*的表达水平(而非组蛋白甲基化水平)实现(Jones et al., 2019)。在转录后水平, 可变剪接可响应温度变化参与的温度补偿过程。当*SKIP*功能缺失后, 17°C低温下, 生物钟周期显著变短, 其长周期表型随着温度的升高逐渐消失(Wang et al., 2012); SIC也参与温度补偿过程(Marshall et al., 2016)。在翻译后水平, 高温下, CK2可磷酸化生物钟的核心组分CCA1, 通过减弱其DNA结合能力参与温度补偿(Portolés and Más, 2010; Marshall et al., 2016)。ELF4的磷酸化也参与温度补偿, 随着温度的升高, *elf4-211* (ELF4^{S45L})突变体的长周期表型则更加显著, 表现出过补偿现象(Choudhary et al., 2015)。SUMO蛋白酶OTS1 (OVERLY TOLERANT TO SALT 1)和OTS2的功能缺失突变体在高温下生物钟周期表型与野生型无差异, 但在低温下周期显著变长, 表现为补偿不足。与之相反, SUMO连接酶SIZ1 (SALT INDUCED ZINC FINGER PROTEIN1)功能缺失后, 在低温下生物钟周期与野生型无差异, 但是在高温下, 周期显著变短, 也表现为补偿不足(Hansen et al., 2017)。不同温度下, 蛋白-蛋白间的互作强度差异也参与温度补偿过程。Cvi背景下, GI^{L718F}和ZTL^{P35T}两个保守位点的共同进化决定了高温下二者的互作减弱, ZTL蛋白水平降低, 从而导致生物钟周期较长(Kim et al., 2020)。

从时间生物学领域看, 温度补偿分子机制研究大致经历了3个阶段: 第1阶段是各种假说概念的提出; 第2阶段是数学和计算机模型的提出, 用于模拟温度补偿曲线; 第3阶段是随着对不同物种生物钟系统核心组分的挖掘及分子网络的建立, 发现可变剪接、蛋白翻译后修饰及不同温度下蛋白亚细胞定位均可参与温度补偿过程, 但目前对温度补偿的理解仍存在不足,

如何利用生物钟的温度补偿特性具有重要研究价值。

4 粮食作物的生物钟组分及相关农艺性状

基于拟南芥生物钟基因在调控代谢和生长发育过程中的重要作用，其同源基因在水稻(*Oryza sativa*)、大豆(*Glycine max*)和玉米(*Zea mays*)等粮食作物中的分子功能吸引了广大研究者的关注，但目前粮食作物的生物钟TTFL网络研究相对滞后。一方面是由于材料自身因素限制，很难通过实时生物荧光节律监测系统筛选及鉴定调控生物钟周期的新基因；另一方面是基于农业生产的目的，大部分研究更关注生物钟基因对重要农艺性状的遗传改良与运用。

目前，利用同源基因比对、全基因组关联分析(GWAS)和图位克隆等方法，在粮食作物中已鉴定到许多与拟南芥同源的生物钟基因，且关于这些生物钟基因所调控的农艺性状，尤其是开花时间和地域适应性方面的分子机制研究取得了重要进展。下面将重点总结现阶段水稻、大豆和玉米等粮食作物中生物钟基因的分子功能及其调控的农艺性状。

4.1 水稻生物钟基因研究现状

水稻是重要的短日照单子叶粮食作物，与拟南芥同源的生物钟基因包括*OsCCA1*、*OsPRR37*、*OsPRR73*、*OsPRR59*、*OsPRR95*、*OsTOC1*/*OsPRR1*、*OsELF3-1*、*OsELF3-2*、*OsELF4a*、*OsELF4b*、*OsELF4c*和*OsGI*等。尽管目前对这些水稻生物钟基因间的互作调控网络并不十分清楚，但大量研究表明它们广泛参与调控水稻的抽穗期、光合作用、作物产量和非生物胁迫等生物学过程(表1)。

OsCCA1 (亦称Nhd1, N-MEDIATED HEADING DATE 1)在调控水稻重要农艺性状方面起至关重要的作用。*OsCCA1*缺失导致水稻抽穗期延迟，一方面，OsCCA1作为转录激活因子，通过激活*OsGI*-*Hd3a* (*HEADING DATE 3A*)信号通路促进开花；另一方面，OsCCA1可作为转录抑制因子，抑制*OsPRR37*/-*DTH8* (*QTL FOR DAYS TO HEADING ON CHROMOSOME 8*)的表达，通过*Ehd1* (*EARLY HEADING DATE 1*)-*Hd3a*信号通路促进开花(Lee et al., 2022)。OsCCA1可以整合糖和独脚金内酯信号调控水稻穗发育和分蘖。糖在根中抑制*OsCCA1*的表达但在茎中促进其表达，OsCCA1则可正调控*OsTB1*和*D14*的表达抑制分蘖。此外，OsCCA1可直接激活*OsAMT1;3* (*AMMONIUM TRANSPORTER1;3*)/*OsNRT2.4* (*high-affinity NITRATE TRANSPORTER 2.4*)的表达，调控水稻根系对铵的吸收和硝的分配。田间低氮条件下，*OsCCA1*突变可提高氮素的吸收和利用效率(Li et al., 2022)。Wei等(2022)研究表明, OsCCA1还正向调控水稻对高盐、干旱及渗透胁迫的适应性，其功能

表1 水稻生物钟基因的功能总结

Table 1 Functional summary of circadian genes in rice

基因名称	拟南芥同源基因	参与的生理过程	参考文献
OsCCA1	*CCA1*	开花时间调控和盐胁迫响应	Lee et al., 2022; Li et al., 2022; Wei et al., 2022
OsPRR1	*PRR1*/*TOC1*	开花时间调控	Li et al., 2023a
OsPRR37	*PRR3*/*PRR7*	开花时间调控	Kwon et al., 2015; Hu et al., 2021
OsPRR73		盐胁迫响应和开花时间调控	Liang et al., 2021; Wei et al., 2021
OsPRR59 *OsPRR95*	*PRR5*/*PRR9*	光合碳固定、开花时间调控、脱落酸和盐胁迫响应	Chen et al., 2022; Wang et al., 2022b
OsGI	*GI*	盐胁迫响应和开花时间调控	Izawa et al., 2011; Wang et al., 2021b
OsELF3-1	*ELF3*	盐胁迫响应和开花时间调控	Zhao et al., 2012; Wang et al., 2021b
OsELF3-2		稻瘟病免疫响应、盐胁迫响应和开花时间调控	Zhao et al., 2012; Ning et al., 2015
OsELF4a *OsELF4b* *OsELF4c*	*ELF4*	盐胁迫响应、开花时间调控和免疫调控	Wang et al., 2021b; Xu et al., 2023
OsLUX	*LUX*	盐胁迫响应和开花时间调控	Wang et al., 2021b

缺失突变体对多种逆境胁迫更加敏感。

*OsPRR37*在调控水稻的纬度适应性方面发挥重要作用, 其变异位点在水稻引种驯化过程中被广泛应用(Koo et al., 2013)。酪蛋白激酶CK1和CK2α能与OsPRR37互作影响其蛋白活性。OsPRR37作为CCT家族蛋白, 主要通过调控*Ehd1*和*Hd3a*的表达来抑制水稻抽穗(Kwon et al., 2015; Hu et al., 2021)。*OsPRR37*的同源基因*OsPRR73*同样具有抑制水稻开花的作用。在长/短日照下, 过表达*OsPRR73*均会导致开花时间延迟。OsPRR73调控开花时间主要通过结合*Ehd1*和*OsCCA1*的启动子, 抑制它们在黎明时表达(Liang et al., 2021)。此外, OsPRR73通过与OsHDAC10的相互作用, 抑制*OsHKT2;1* (*HIGH-AFFINITY K^+ TRANSPORTERS2;1*)的转录, 调节细胞内Na^+/K^+稳态, 从而增强对盐胁迫的耐受性(Wei et al., 2021)。Chen等(2022)研究表明, OsPRR59和OsPRR95直接参与调控光合作用, 其表达也受到光周期调控。长日照下, OsPRR59可直接结合*Ehd3*的启动子抑制其表达, 进而延迟水稻抽穗。此外, 其还通过影响株高、二级枝梗数、每穗粒数、结实率和单株粒重正调控产量(Wang et al., 2022b)。OsTOC1既可作为转录激活子又可作为转录抑制子, 通过调控不同的靶基因控制生物钟输出途径。过表达*OsTOC1*导致水稻抽穗延迟, *OsTOC1-OE*植株还表现出比野生型更多的分蘖数量。另外, Li等(2023a)研究表明, 与野生型相比*OsTOC1-OE*植株表现出更强的病原菌抗性。

水稻包含2个ELF3的同源蛋白OsELF3-1和OsELF3-2, 3个ELF4的同源蛋白OsELF4a、OsELF4b和OsELF4c。在水稻栽培过程中, *OsELF3-1*受到人工选择, 对水稻抽穗期的控制作用更为显著。短日照下, OsELF3-1通过激活*Ehd1*使水稻抽穗期提前, 但长日照下其反而作为抽穗抑制因子发挥作用(Zhao et al., 2012)。OsELF3-2主要参与水稻抗病性调控(Zhao et al., 2012; Ning et al., 2015)。OsELF4s为长日照下调控水稻抽穗期所必需, 但相关分子机制并不清楚。OsEC1是一种由OsELF4a、OsELF3-1和OsLUX组成的蛋白复合物, 可直接结合到*OsGI*的启动子上抑制其表达。OsGI一方面通过调控*Hd1*的表达影响抽穗时间; 另一方面通过调控下游Na^+以及K^+相关转运体基因*OsHKT2;1*/*2;3*/*2;4*的表达抵御盐胁迫。Wang等(2021b)研究表明, OsEC1也可能通过抑制*OsPRR73*和*Hd1*的表达负调控水稻抽穗期, 该调控途径独立于OsGI通路。*OsGI*作为OsEC1平衡抽穗期和盐胁迫响应的靶基因, 其突变体在长日照下表现出提早抽穗以及耐盐胁迫表型(Izawa et al., 2011; Wang et al., 2021b)。此外, OsGI还可与水稻特有的开花调控因子Ghd7 (GRAIN NUMBER、PLANT HEIGHT和HEADING DATE 7)相互作用, 促进其降解, OsPHYA (PHYTOCHROME A)和OsPHYB则抑制OsGI与Ghd7蛋白互作, 从而稳定Ghd7蛋白(Zheng et al., 2019)。

4.2 大豆生物钟基因研究现状

大豆是典型的四倍体、短日照、双子叶经济作物, 其开花时间对光周期极其敏感, 适合在高纬度地区种植。将北方的大豆引种到南方栽培时, 成熟期会显著提前, 导致大豆产量和生物量均显著降低。*ELF3*在大豆中的同源基因*J* (*JUVENILE*)可提高大豆对低纬度地区的适应性(表2)。*J*等位基因的功能缺失导致大豆营养生长期显著延长, 使大豆在开花前能获得更多的光合产物, 突破了大豆在低纬度地区产量极低的限制。这也促使大豆在南方低纬度地区得以推广(Lu et al., 2017)。*LUX*的同源基因在大豆开花时间调控中同样发挥重要作用, 缺乏*GmLUX1*和*GmLUX2*的大豆植株在长/短日照下均表现出极端晚花表型, 二者双突变会导致大豆丧失对光周期变化的识别能力(Bu et al., 2021), 表明生物钟EC可能是大豆光周期现象的核心调控因子。

J是大豆开花抑制因子E1的直接转录抑制因子, J蛋白可结合在*E1*的启动子上抑制其转录, 从而解除对2个开花基因*FT2a* (*FLOWERING LOCUS T 2a*)和*FT5a*的抑制, 进而促进大豆在短日照下开花(Lu et al., 2017)。*GI*在大豆中的同源基因*E2*、*E2La*和*E2Lb*也被发现功能冗余地参与调控大豆开花时间和产量形成。高海拔地区, *e2*单突变体呈现早花且高产的农艺性状(Wang et al., 2023)。近年来陆续发现许多参与调控大豆EC蛋白稳定性和转录活性的因子。Liu等(2008)研究表明, 大豆基因组共编码4个PHYA, 其中GmPHYA2 (E4)和GmPHYA3 (E3)是光周期开花调控途径的抑制因子, 分别介导高红光和远红光下的大豆开花过程。E3/E4可直接与LUX蛋白互作, 降低其稳定性, 促进*E1*表达进而导致晚花(Lin et al., 2022)。此外, Qin等(2023)研究表明, 光下激活的E3/E4与

表2 大豆生物钟基因的功能总结

Table 2 Functional summary of circadian genes in soybean

基因名称	拟南芥同源基因	参与的生理过程	参考文献
E2	*GI*	开花时间调控和产量形成	Dong et al., 2022; Wang et al., 2023
E2la			
E2lb			
GmLHY1a	*LHY*	干旱胁迫响应和开花时间调控	Marcolino-Gomes et al., 2014; Li et al., 2019; Wang et al., 2020b; Dong et al., 2021; Yuan et al., 2021b
GmLHY1b			
GmLHY2a		开花时间调控	
GmLHY2b			
GmLUX1	*LUX*	开花时间调控	Bu et al., 2021
GmLUX2			
GmELF3/J	*ELF3*	开花时间调控和盐胁迫响应	Lu et al., 2017; Cheng et al., 2020
GmPRR3a	*PRR3*	干旱胁迫响应和开花时间调控	Syed et al., 2015; Lu et al., 2020
GmPRR3b			

GmEID1互作，从而抑制其与J蛋白的相互作用，促进J蛋白降解。近期，研究发现由GmFKF1/GmGI(E2)-EC组成的“太极阴阳平衡”对于大豆光周期感知至关重要。白天，蓝光激活GmFKF1与E2的互作，靶向J蛋白并促进其降解。夜晚，由于GmFKF1与E2解离，J蛋白逐渐积累并与LUX结合形成足够的EC复合物，该复合物反过来结合在*E2*的启动子区域抑制其表达(Zhao et al., 2024)。长日照下，J蛋白大量降解减轻了其对E1的抑制，进一步促进*E1*和*E1Lb*的表达。短日照下，由于E3和E4功能减弱，同时GmFKF1可能无法与E2形成足够的复合物降解J蛋白，造成J蛋白在短日照下大量累积，因此*E1*的表达水平很低，导致大豆提早开花(Zhao et al., 2024)。

*PRR3*在拟南芥中的研究相对较少，但其同源基因*Tof11* (*TIMING OF FLOWERING 11*)和*Tof12*对于促进大豆向高纬度地区种植发挥重要作用。研究表明，E3/E4可促进*PRR3a* (*Tof11*)和*PRR3b* (*Tof12*)的表达，PRR3a/PRR3b则可抑制*GmLHYs*/*GmCCA1s*的表达(Lu et al., 2020)。与J蛋白功能相似，由*GmLHY1a*编码的Tof16位点被证实同样有助于大豆适应低纬度地区，但该过程独立于J蛋白。短日照下，*GmLHY1a*和*GmLHY1b*功能缺失突变体显著提高大豆产量并延迟开花。*GmLHY1a*也通过直接结合并抑制*E1*的表达来介导光周期开花调控(Dong et al., 2021)。

除开花时间调控外，Cheng等(2020)发现NaCl处理也诱导*J*的表达。盐胁迫下，J蛋白可通过正向调控下游胁迫响应基因的表达调控耐盐性。大豆*E2*对作物产量和盐胁迫均有负调控作用。*e2*单突变体在高纬度地区的开花时间提前，同时产量提高(Wang et al., 2023)。盐胁迫后，*e2*突变体通过释放过氧化物酶和清除活性氧(ROS)表现出耐盐表型(Dong et al., 2022)。干旱胁迫降低了大豆生物钟基因的表达，包括*LCL1*、*GmELF4*和*PRRs*类基因(Marcolino-Gomes et al., 2014; Li et al., 2019)。大豆*LHY*/*CCA1*同源基因*GmLCLa1*、*GmLCLa2*、*GmLCLb1*和*GmLCLb2*在干旱胁迫响应中具有重要作用。干旱胁迫下，*GmLCLs*四突变体的叶片失水速率比野生型更低，干旱胁迫也延迟了*GmLCLs*基因的峰值表达，表明GmLCLs与干旱胁迫间存在复杂的反馈关系(Yuan et al., 2021b)。此外，干旱胁迫下，*GmLCLs*四突变体ABA信号相关基因的表达量升高，如*PYL17* (*PYRABACTIN RESISTANCE-LIKE 17*)、*CYP707A* (*CYTOCHROME P450 707A*)、*ABI2* (*ABA INSENSITIVE 2*)和*SnRK2s* (*SNF1-RELATED PROTEIN KINASE2s*)，以应对干旱胁迫(Wang et al., 2020b; Yuan et al., 2021b)。Syed等(2015)研究表明，干旱条件下，*GmPRR3*基因还会发生可变剪接。GmPRR3可与GmPRR7和GmTOC1协同作用，实现干旱胁迫响应。

综上，大豆生物钟系统组分也广泛参与调控光周期依赖的开花、作物产量及非生物胁迫响应过程，但生物钟系统如何协同光温信号调控大豆的生长发育过程仍需深入探究。

4.3 玉米生物钟基因研究现状

玉米是二倍体、短日照、单子叶粮食作物, 起源于墨西哥南部, 现已在全球范围内广泛种植。玉米从热带向温带地区扩张伴随着其光周期敏感性降低。近年来, 关于玉米生物钟系统的研究逐步展开, 且已发现许多与拟南芥同源的候选基因, 如*ZmCCA1*、*ZmLHY*、*ZmTOC1*、*ZmPRR73*、*ZmPRR59*、*ZmGI*和*ZmFKF1* (表3)。

ZmELF3.1和ZmELF3.2蛋白可与ZmELF4.1/4.2及ZmLUX1/2相互作用, 形成玉米的晚间复合物EC。ZmELF3.1/3.2及ZmLUX1/2的功能缺失突变体在长/短日照下均表现出开花延迟表型。Zhao等(2023)研究表明, EC可直接抑制开花抑制基因(如*ZmPRR37a*和*ZmPRR73*)的表达, 从而削弱其对下游成花素基因表达的抑制作用, 以间接促进开花。目前, 在玉米热带系CML288中鉴定出3个*ZmCCA1*的可变剪接体, 即*ZmCCA1.1*、*ZmCCA1.2*和*ZmCCA1.3*, 其分别编码3种不同的蛋白亚型。干旱胁迫诱导*ZmCCA1*的表达, 可增强玉米对干旱胁迫的耐受性。Tian等(2019)研究表明, *ZmCCA1.1*可能是抗旱的显性基因, 而*ZmCCA1.2*和*ZmCCA1.3*的作用相对较小。*GI*在玉米中有2个同源基因, 其中Zm*GI1*的表达量较高。长日照下, *gi1*突变体早花, 但在短日照下其开花时间与野生型相比无差异。此外, *ZmGI1*还控制玉米从营养生长向生殖生长的转变及株高等农艺性状(Bendix et al., 2013)。

综上, 越来越多的农作物生物钟系统被发现广泛参与调控光周期依赖的开花、作物产量及非生物胁迫响应过程, 未来有望应用生物钟系统精细调控作物的产量性状。

5 研究展望

输入途径、核心振荡器和输出途径是植物生物钟系统3个密切联系、不可分割的部分。此3部分的正常运转可使植物感知和预测昼夜性和季节性环境变化, 优化资源利用(如白天进行光合作用获取能量, 夜间进行物质合成与储备), 从而在适宜的时间生长、发育、开花和结实, 对于植物适应生存环境具有重要意义。

早期生物钟系统的研究主要集中在核心振荡器组分挖掘、功能分析及转录-翻译反馈环的建立。目前, 对核心振荡器关键组分转录-翻译反馈环的研究已较为成熟, 并在转录后、翻译后及表观修饰层面取得了重要进展, 但仍有许多问题亟待解决。

(1) 转录后层面, 许多生物钟基因mRNA上存在m^6A修饰, 但对于mRNA命运的调控机制及其功能尚缺乏系统的研究。另外, m^6A修饰为高度动态的过程, 目前的研究表明m^6A甲基转移酶参与调控生物钟周期, m^6A去甲基化酶是否及如何影响生物钟并不清楚。此外, 大部分生物钟基因都存在不同形式的可变剪接体, 但对所产生的蛋白异构体的功能及其对周期的影响研究较少。

表3 玉米生物钟基因的功能总结

Table 3 Functional summary of circadian genes in maize

基因名称	拟南芥同源基因	参与的生理过程	参考文献
ZmGI1	*GI*	开花时间调控和株高控制	Bendix et al., 2013; Li et al., 2023b
ZmGI2		开花时间调控	
ZmPRR1-2	*PRR1*/*TOC1*	开花时间调控	董柯清等, 2023
ZmELF3.1	*ELF3*	开花时间调控	Zhao et al., 2023
ZmELF3.2			
ZmELF4.1	*ELF4*	开花时间调控	Zhao et al., 2023
ZmELF4.2			
ZmLUX1	*LUX*	开花时间调控	Zhao et al., 2023
ZmLUX2			
ZmPRR37a	*PRR3*/*PRR7*	开花时间调控	Zhao et al., 2023
ZmPRR73			
ZmCCA1	*CCA1*	干旱胁迫响应	Tian et al., 2019

(2) 翻译后层面，尽管PRR7/PRR9与TOC1/PRR5高度同源，但是ZTL家族成员并不介导二者的泛素化降解，ZTL特异性降解TOC1/PRR5而非PRR7/PRR9的原因是什么？PRR7/PRR9的降解机制又是什么？PRR7蛋白也存在高度动态的磷酸化修饰，但相应的蛋白激酶尚未被鉴定。再有，不同时间点的翻译后修饰位点是否不同？功能是否存在差异？

(3) 表观修饰层面，核心生物钟组分启动子区域的表观修饰变化趋势通常与基因表达时相一致或相反，即也呈现动态的节律性变化。那么负责表观修饰的蛋白酶的节律如何被生物钟调控？

(4) 宏观角度上，目前对拟南芥生物钟系统TTFL的理解主要基于常温下幼苗叶片的分析。然而根据已有研究，生物钟周期在顶端分生组织、叶片和根部具有明显的组织特异性。Chen等(2020)研究表明，ELF4可从茎向根部移动，从而调节根部的生物钟节律，且ELF4移动的快慢受温度调节。那么，不同组织部位的生物钟信息交流是否均依赖ELF4？此外，不同生物钟基因的组织表达模式说明，它们会在不同的组织高度富集，暗示在不同的组织部位、发育时期和温度下，核心振荡器转录-翻译反馈环可能会发生相应的变化。

近年来，随着全球气候变化，越来越多的研究开始关注环境光、温信号到生物钟的输入过程。全面解析光、温信号驯化生物钟的分子网络，对科研人员理解和预测环境光、温信号变化调控内源生物钟基因的表达节律机制，从而影响生长发育的过程至关重要。首先，短日照植物(如水稻和大豆)的开花时间对光周期特别敏感，这限制了其不同品种的地域分布。解析这些作物响应光周期的分子机制，可在特定的时间段，通过改变补光时间、光质或光强，改变参与识别光周期的生物钟基因(如*GI*和*FKF1*)的表达相位，实现人为调控开花时间。其次，农作物生长发育的苗期、授粉期及灌浆期对温度极为敏感，而寒潮和高温现象频发对农作物发育和稳产是巨大的挑战。生物钟可门控温度信号，“关闭”植物对高温或低温特别敏感的时间段(促进或抑制温度响应基因的表达)，进而有效缓解温度胁迫对植物的伤害。再有，根据温度补偿特性，生物钟也可缓冲温度波动对生物钟周期造成的干扰，对于维持细胞内代谢平衡及适应温度变化具有重要意义。解析生物钟维持温度补偿的分子机制，有助于利用基因编辑技术培育耐温度胁迫且不影响生长发育的材料，对农业育种具有潜在的应用价值。

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Advances of Plant Circadian Clock Response to Light and Temperature Signals

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Abstract With the sharp change of the global climate, the ecological environment for plant is becoming increasingly harsh, therefore the molecular mechanisms underlying how circadian synergistically interacts with light or temperature receptors to transmit environmental signals and rhythmically regulate various growth and development process received widespread attention. As an endogenous timer of plants, the core oscillator of circadian clock is composed of multiple coupled transcriptional-translational feedback loops (TTFL), and it is modified from transcription, post-transcription, translation, post-translation to epigenetic levels. These multi-precise regulatory mechanisms ensure that the circadian clock can be synchronized and reset by external signals, so that the endogenous rhythm matches with external cycles, thereby endowing plants with the ability to optimize resource utilization and tend towards the optimal growth, which also has an important significance for guiding the genetic improvement and domestication of crops. In this review, we summarized the multi-level of regulatory mechanisms of core oscillator as well as the molecular function of circadian homologous genes in crops, thoroughly described the interaction network between the circadian clock and the light and temperature signal pathways and give prospects for molecular breeding based on the opinion, which provides new ideas for expanding the environmental adaptability and optimizing agronomic traits of crops.

Key words circadian clock, light and temperature signals, TTFL, environmental adaptability, molecular breeding

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