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·专题论坛·

转录因子在植物响应UV-B辐射中的调控作用

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摘要 作为太阳光的固有成分，UV-B对植物生长发育有重要影响。随着对UV-B研究的深入，人们认识到UV-B不仅是环境胁迫因子，还是植物生长过程中的重要信号分子，适度的UV-B辐射对植物生长具有促进作用。UVR8是UV-B特有的光感受器，在植物响应UV-B过程中发挥不可替代的作用，且其功能受上、下游转录因子调节。目前，已知BBX、WRKY、MYB和PIF等多种转录因子参与调控UV-B辐射下的下胚轴伸长、主根长度、叶片大小及形态、开花周期和花青素合成等过程。该文主要综述了UVR8在UV-B信号通路中的分子机制，并对转录因子在UV-B辐射过程中的调控机理进行总结，以期为相关研究提供参考。

关键词 UVR8，转录因子，UV-B辐射，植物

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太阳光是植物生长发育过程中重要的环境因子。UV-B (ultraviolet radiation B, 275–320 nm)是太阳光中的固有成分，直接影响植物的生长发育。因此，自然环境中的UV-B被视为一种潜在的影响植物生长的环境信号(Hayes et al., 2014; 陈慧泽和韩榕, 2015; Shi and Liu, 2021)。作为UV-B特定的光感受器, UVR8 (UV RESISTANCE LOCUS 8)被UV-B激活后，通过调节下游基因的表达调控UV-B光形态的发生，如植物的主根变短、侧根数量增多及下胚轴缩短(Kim et al., 1998; Qian et al., 2016; Yadav et al., 2020)。其发挥作用依赖于不同的转录因子家族，包括bZIP (BASIC LEUCINE ZIPPERS)、MYB (V-MYB AVIAN MYELOBLASTOSIS VIRAL ONCOGENE HOMOLOG) 和 BBX (B-BOX DOMAIN PROTEIN) 等(Binkert et al., 2014; Yadav et al., 2020; Yang et al., 2020; Xu and Zhu, 2020)。

转录因子是一种DNA结合蛋白，在植物中普遍存在，其通过复杂的转录网络调控大量基因的表达，使植物维持正常的生长发育，是植物适应环境的基础(Weidemüller et al., 2021; Strader et al., 2022)。转录因子可调节植物早期胚胎发育和种子成熟过程，控制生长素响应基因的表达，参与开花时间调控以及生物和非生物胁迫响应，通过各种复杂的转录网络调控植物生长发育和环境适应能力(Ratcliffe and Riechmann, 2002; Guilfoyle and Hagen, 2007; Ng et al., 2018; Verma et al., 2022)。

转录因子在光信号通路中发挥至关重要的作用。近年来，随着对转录因子研究的深入，已发现越来越多的转录因子在UVR8介导的UV-B信号通路中发挥重要作用(刘明雪等, 2012; Liang et al., 2018; 陈慧泽等, 2021; Fang et al., 2022)。UVR8可通过直接与细胞核中的转录因子BIM1/BES1 (BRI1-EMS-SUPPRESSOR1/BES1-INTERACTING MYC-LIKE 1)和WRKY36 (WRKY DNA-BINDING PROTEIN 36)相互作用介导UV-B信号转导；也可通过COP1 (CONSTITUTIVE PHOTOMORPHOGENIC 1)调节转录因子HY5 (ELONGATED HYPOCOTYL 5)的蛋白稳定性，从而诱导下游光响应基因的表达(Favory et al., 2009; Huang et al., 2012; Liang et al., 2018; Yang et al., 2018)。据报道，锌指转录因子BBX家族中很多成员参与调控UV-B信号转导。其中, *BBX24* (*B-BOX* DOMAIN PROTEIN 24)是UV-B光形态建成的负调控

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因子; *BBX31*则是UV-B光形态建成的正调控因子。在UV-B辐射下，BBX20–22作为HY5的辅助调节因子，与HY5共同调节其自身的转录活性。还有其它成员也参与响应UV-B辐射(Jiang et al., 2012; Yadav et al., 2019a; Lyu et al., 2020) (表1)，但其作用的具体分子机制尚不清楚。本文主要对转录因子参与调控UV-B信号通路的分子机制进行概述。

1 UV-B辐射的主要信号通路

UVR8光感受器是UV-B信号通路中的关键信号分子，植物体内由UV-B诱导的光形态建成以及生物色素合成主要通过UVR8介导的信号通路完成(Haves et al., 2017)。研究表明, UVR8响应UV-B主要有2种不同的分子机制。一是UVR8与E3泛素连接酶COP1结合促进转录因子HY5的积累，通过调节转录因子的活性，从而调控UV-B诱导的基因表达；另一种是UVR8直接与转录因子相互作用，调控UV-B光响应基因的表达(Binkert et al., 2014; Yin et al., 2016; Liang et al., 2018, 2019; Xu and Zhu, 2020; Wang et al., 2022; Zhang et al., 2022)。

1.1 UVR8-COP1介导的UV-B信号转导

E3泛素连接酶COP1作为光形态建成的抑制因子，其C端的WD重复结构域对于行使功能至关重要。该结构域可识别底物、结合DDB1 (DAMAGE-SPECIFIC DNA BINDING PROTEIN 1)及与多种光感受器相互作用，在COP1的底物中VP基序被鉴定为与COP1相互作用的位点。UVR8在植物体内以二聚体的形式存在，当植物受到UV-B辐射时，UVR8被激活并解聚成单体进入细胞核，通过其C端的VP基序与COP1的WD40结构域互作，进行UV-B信号转导，且COP1还包含NLS信号和核输出信号，是UVR8核积累所必需，因此COP1是UVR8信号通路的重要组分(Oravecz et al., 2006; Yin et al., 2016; Rai et al., 2019; Zhang et al., 2023)。此外, COP1蛋白C端的WD40结构域还可与多种促进光形态建成的转录因子互作，并介导这些转录因子进入泛素化降解，从而调控下游基因的表达

表1 转录因子在植物响应UV-B信号通路中的作用

Table 1 Function of transcription factors in plant response UV-B radiation

家族	成员	顺式元件	作用	参考文献
bZIP	HY5	T/G-box、E-box、GATA-box、ACE-box、Z-box和C-box	调节下胚轴伸长，在植物色素合成和光形态建成中起核心作用	Stracke et al., 2010a; Lockhart, 2014; Yao et al., 2021; Zhou et al., 2022b
WRKY	WRKY36	W-box	促进下胚轴伸长及叶片发育	Yang et al., 2018; Demarsy, 2022; Goyal et al., 2023
	WRKY32	W-box	促进下胚轴伸长，抑制拟南芥子叶展开	Zhou et al., 2022a; Demarsy, 2022
MYB	MYB11、MYB12和MYB111	R2R3-MYB-binding motif	抑制下胚轴伸长，调控花青素生物合成，影响次生代谢物合成	Stracke et al., 2007, 2010b; Petroni et al., 2008; Zhang et al., 2017; Wang et al., 2021b; Zhao et al., 2023
	MYB73/77	R2R3-MYB-binding motif	正调控生长素响应基因的表达，促进下胚轴伸长和侧根生长	Yang et al., 2020; Xu and Zhu, 2020; Yang and Liu, 2020; Zhang et al., 2022
BBX	BBX11	T/G-BOX	调节拟南芥下胚轴伸长，参与植物光形态建成	Job et al., 2022; Ponnu, 2022
	BBX20、BBX21、BBX22和BBX31	T/G-BOX	光形态建成正调控因子	Heng et al., 2019; Bursch et al., 2020; Podolec et al., 2022
	BBX24	T/G-BOX	光形态建成负调控因子	Jiang et al., 2012; Gangappa and Botto, 2014
PIF	PIF4和PIF5	G-box	抑制下胚轴伸长，参与植物光形态建成	Sharma et al., 2019; Küpers et al., 2020; Podolec et al., 2021; Zhao et al., 2023; Sharma et al., 2023
TCP	TCP4	GGACCAC	调控植物生长发育，参与光响应	Li et al., 2024
BES	BES1/BIM1	E-box和CGTGT/CG	调节植物激素信号转导，影响植物生长发育	Liang et al., 2020; Shi and Liu, 2021

(Oravecz et al., 2006; Podolec and Ulm, 2018; Sharma et al., 2019; Yadav et al., 2020; Han et al., 2020)。

转录因子HY5可直接结合光信号响应元件T/G-box等多种顺式作用元件，调节下游基因的表达，已确定其是UV-B诱导基因表达的正调控因子，在UV-B信号转导中起核心作用。HY5也是COP1-SPA (CONSTITUTIVE PHOTOMORPHOGENIC 1-SUPPRESSOR OF PHYA-105)复合物降解的第1个目标底物，且含有VP基序(Gangappa and Botto, 2016; Wang et al., 2021a)。COP1通过与HY5的VP基序结合抑制其积累，还使HY5同源蛋白HYH (HY5 HOMOLOG)降解(Brown et al., 2005; Yadav et al., 2020; Wang et al., 2021a; Zhang et al., 2022)。而在UV-B作用下，解聚为单体的UVR8与细胞核内的COP1结合，以减少COP1对HY5的降解，从而使HY5的转录活性增强，进而调节下游UV-B光响应基因的表达。HY5在UV-B下的积累也可通过RUP1 (REPRESSOR OF UV-B PHOTOMORPHOGENESIS 1)和RUP2的积累来平衡，RUP2还直接与COP1相互作用，抑制COP1与HY5的结合(Brown et al., 2005)。

此外，UV-B光形态建成抑制因子RUP1和RUP2也含有WD40结构域，并直接与UVR8相互作用。与COP1不同的是，RUP1和RUP2仅含有WD40重复序列，它们的丰度变化可能影响整个复合物的活性或特异性，而*COP1*的表达是组成型的，不受UV-B调节，因此，*RUP1*和*RUP2*的表达对植物适应光环境变化至关重要(Gruber et al., 2010; Chen et al., 2024)。UVR8在UV-B的诱导下解聚成单体与COP1相互作用，而RUP1和RUP2可使UVR8重新聚合为二聚体，导致UVR8与COP1的互作被抑制，从而在UV-B信号转导中起负调节作用，且在这个过程中它们的激活需要UVR8、COP1和HY5的参与(Gruber et al., 2010; Heijde and Ulm, 2012; Fang et al., 2022; Wang et al., 2023a)。

1.2 UVR8与转录因子互作调控基因表达

UVR8直接与转录因子结合调节下游基因的表达也是UV-B信号转导的重要途径。白光下，转录因子BES1/BIM1定位于细胞核，不能被UVR8诱导；而在UV-B辐照下，UVR8在细胞核中与BES1/BIM1相互作用，抑制其DNA结合活性(Liang et al., 2018, 2020; Shi and Liu, 2021)。转录因子MYB73/MYB77正调控生长素响应基因的表达，促进下胚轴伸长和侧根生长，被UV-B激活的UVR8在细胞核中与MYB73/MYB77直接互作，抑制其DNA结合活性，导致MYB73/77与IAA19解离，抑制生长素响应基因的表达(Yang et al., 2020; Xu and Zhu, 2020; Yang and Liu, 2020; Zhang et al., 2022)。另一种MYB转录因子MYB13由UV-B诱导，主要在子叶中表达，其可与生长素响应基因*SAUR* (*SMALL AUXIN UPREGULATED RNA*)启动子结合调控子叶膨胀，解聚成单体的UVR8可促进MYB13与*CHS* (*CHALCONE SYNTHASE*)和*CHI* (*CHALCONE ISOMERASE*)的启动子结合，增强植物对UV-B胁迫的抗性；也导致MYB13与*SAUR*分离，抑制*SAUR*的表达；但随着UV-B辐照时间的延长，MYB13可能在不受UVR8控制的情况下积累，从而激活*SAUR*，促进子叶的扩张(Hayes et al., 2014; Qian et al., 2020; Xu and Zhu, 2020)。此外, UVR8可以与转录因子TCP4 (TEOSINTE BRANCHED1, CYCLOIDEA, PROLIFERATING CELL FACTOR 4)直接相互作用，增强TCP4的DNA结合活性，从而激活JA (jasmonate)生物合成基因*LOX2* (*LIPOXYGENASE2*) (Li et al., 2024)。综上, UVR8结合转录因子既可激活其活性，增强下游基因的表达，也可抑制其DNA结合域，进而阻断下游基因的表达，具体作用取决于UVR8结合转录因子的不同结构域。

2 转录因子HY5是UV-B信号通路的中心枢纽

bZIP转录因子HY5是UVR8和COP1的调节因子，其可直接结合DNA并在多个位点上调控大量UV-B光响应基因的表达，在植物感应UV-B辐照后快速做出响应，是调节UV-B光形态建成的中心枢纽(Stracke et al., 2010a; Lockhart, 2014; Yao et al., 2021; Zhou et al., 2022b)。HY5通过调节下胚轴伸长及花青素合成等途径促进植物适应UV-B环境(Brown et al., 2009; Saini et al., 2020; Miao et al., 2021; Li et al., 2023)。已证实在拟南芥(*Arabidopsis thaliana*)及番茄(*Solanum lycopersicum*)等植物中HY5可直接调控花青素合成相关基因的表达，还通过诱导MYB转录因

子的转录调控花青素等生物色素合成相关基因的表达，进而参与花青素的合成(Dubos et al., 2010; Stracke et al., 2010a; Shin et al., 2013)。在UV-B辐照下，除HY5自身被激活外，其同源蛋白HYH也可结合在*HY5*的启动子，激活*HY5*的转录活性。在调节UV-B诱导的*HY5*转录激活过程中，*HY5*和*HYH*具有功能冗余。此外，WRKY、MYB和BBX等转录因子也调节*HY5*的转录活性(Binkert et al., 2014; Xu, 2020; Zhang et al., 2022b)。

2.1 UVR8与WRKY36结合稳定*HY5*的转录活性

与抗逆相关的植物WRKY转录因子家族为特有的锌指蛋白转录因子，也是植物中最大的转录因子家族之一，可通过与*HY5*的启动子结合抑制其转录活性，进而促进下胚轴的伸长(Yang et al., 2018; Demarsy, 2022; Goyal et al., 2023)。除UVR8、COP1和HY5等主要成员之外，WRKY36是在UV-B信号通路中新发现的成员。在白光下，UVR8主要定位于细胞质，而转录因子WRKY36定位于细胞核，并直接与*HY5*中的W-box结合进而抑制*HY5*的表达；在UV-B辐射下，UVR8被激活并进入细胞核，直接与WRKY36相互作用，抑制其转录活性，进而促进*HY5*的转录和表达，*WRKY36*作为*HY5*的负调节因子参与UV-B信号转导(Yang et al., 2018; Liang et al., 2018; Zhang et al., 2022)。WRKY家族的另一个成员WRKY32在光形态建成中也发挥积极的调控作用。$WRKY32^{P409L}$可促进下胚轴伸长，抑制拟南芥子叶展开，它作为一种新底物在COP1的下游发挥作用；此外，$WRKY32^{P409L}$还可以直接与*HY5*的启动子结合并在转录水平上正调控*HY5* (Zhou et al., 2022a; Demarsy, 2022)，但它是否响应UV-B及其作用机制仍不清楚。

2.2 HY5通过调节MYB转录提高植物抗UV-B能力

MYB家族是真核生物中最大的转录因子家族，其中R2R3-MYB家族蛋白参与植物的生长发育、生理代谢、细胞分化、器官发育及生物/非生物胁迫响应等(Dubos et al., 2010; Cao et al., 2020; Yin et al., 2021; Wang et al., 2023b; Cai et al., 2023)。在受到高剂量UV-B辐射时，植物会通过增加黄酮类物质及羟基肉桂酸衍生物等的积累来提高抗UV-B能力。研究表明，MYB转录因子参与植物花青素的生物合成，其中*MYB11*、*MYB12*和*MYB111*是幼苗中花青素合成的主要调节基因且在功能上相似(Petroni et al., 2008; Yan et al., 2021; Li et al., 2022; Wang et al., 2023b)。在幼苗早期，*MYB12*主要在根中表达，*MYB111*在子叶中表达，*MYB11*在整个幼苗期的表达水平都非常低，且在白光和UV-B辐照下，HY5是它们被激活所必需(Stracke et al., 2007, 2010a; Petroni et al., 2008; Wang et al., 2021b)。HY5和MYB12协同调节*CHS*等基因的表达，在早期UV-B辐照中，HY5结合在*MYB12*的启动子区对UV-B做出响应；随着UV-B辐照时间的延长，HY5与MYB12结合共同调节下游*CHS*等基因的表达，提高植物对UV-B的耐受性(Stracke et al., 2010a; Lin et al., 2021)。

2.3 锌指转录因子BBX通过HY5参与调控UV-B信号转导

BBX (B-box)蛋白是锌指结构蛋白家族的一个亚家族，该家族许多成员参与UV-B响应。在这个过程中，BBX蛋白与HY5转录因子相互作用形成一个复杂的调控网络；或作为HY5的辅助调节因子，增强其对下游基因的转录调控，从而调节植物的光形态建成(Gangappa and Botto, 2014, 2016; Xu, 2020; Yang et al., 2022)。

*BBX24*是光形态建成的负调控因子，UV-B可诱导其表达和积累。在UV-B信号通路中，UVR8与COP1结合进而增加BBX24的积累，BBX24可直接与HY5结合抑制其转录活性，从而抑制其对下游靶基因的调控，它在UV-B信号通路中与HY5有拮抗作用，也是UV-B光形态建成的负调控因子(Jiang et al., 2012; Gangappa and Botto, 2014)。*BBX31*是白光下光形态建成的负调控因子，白光下其独立于HY5调控光形态建成，但是它在UV-B辐照下的调控作用则依赖于HY5 (Heng et al., 2019)。在UVR8的介导下，HY5可以直接结合在*BBX31*的启动子上并调节其转录，且BBX31正调节*HY5*的转录，与*BBX24*相反，*BBX31*在UV-B信号通路中是正调控因子，在UV-B辐照下，过表达*BBX31*可通过促进类黄酮等代谢产物的积累对幼苗起保护作用(Yadav et al., 2019a, 2019b)。*BBX11*也调控UV-B下的光形态建成，在低剂量UV-B辐射下*BBX11*可调节拟南芥下胚轴的伸长，在高剂量UV-B辐射下可通过调节抗氧化物质的积累提高植

物抗UV-B的能力; 且它在UV-B信号通路中受到*HY5*的正向调控, 还可通过调节*HY5*的转录活性, 与HY5形成*HY5-BBX11*负反馈回路以调节UV-B响应(Job et al., 2022; Ponnu, 2022)。BBX20–22可调节*HY5*的转录活性, 经UV-B短暂辐照的拟南芥中*HY5*的转录活性不依赖于BBX20、BBX21和BBX22; 但延长UV-B辐照时间后, *HY5*则依赖于它们作为共转录激活因子(Podolec et al., 2022)。HY5可负调控BBX21以及BBX22的稳定性。此外, BBX22可结合*HY5*, 并与BBX20和BBX21共同促进光形态建成(Datta et al., 2008; Bai et al., 2014; Bursch et al., 2020)。

2.4 PIFs与HY5分别在UV-B信号通路的2个分支中发挥重要作用

bHLH转录因子家族的光敏色素互作因子PIFs (PHYTOCHROME-INTERACTING FACTORS)与HY5分别在光信号通路的2个分支中作为中间体发挥重要作用(Gaillochet et al., 2020; Yadukrishnan and Datta, 2021)。PIF转录因子在黑暗中稳定而在光照下降解, 是光形态建成的负调控因子; 而与其拮抗的HY5则在

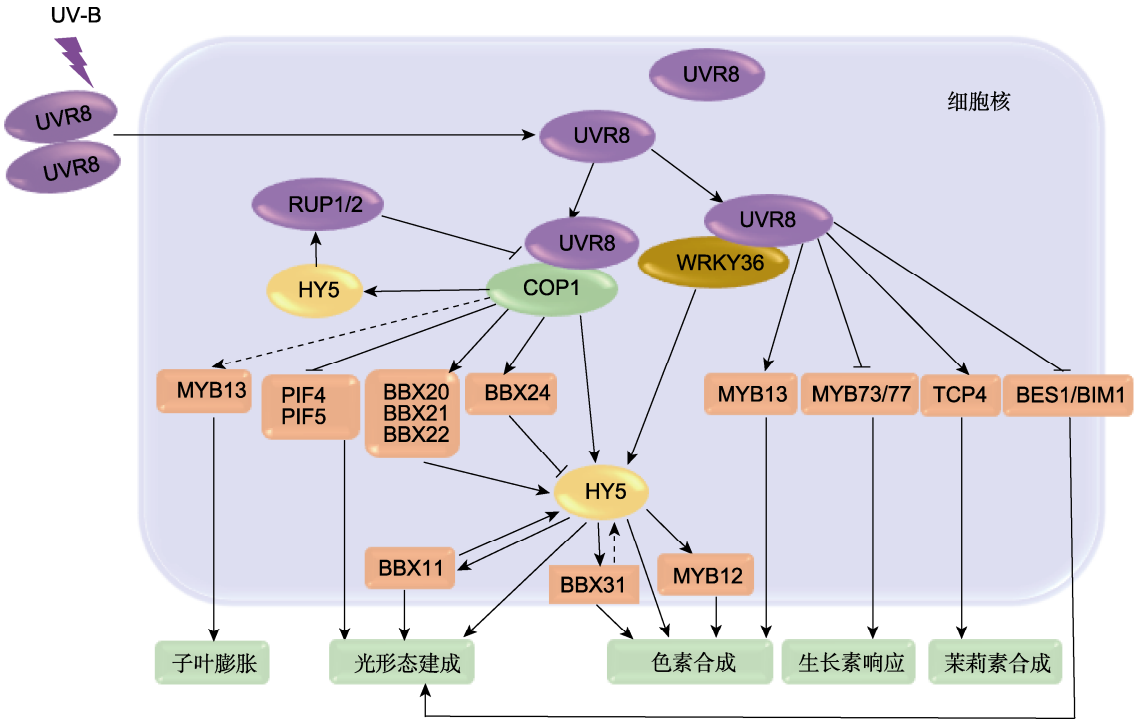


图1 转录因子参与植物UV-B信号通路模型

紫外线B (UV-B)激活其受体UVR8后, UVR8解聚为单体进入细胞核, 进而引发下游信号通路。UVR8直接与E3泛素连接酶COP1作用, 释放COP1降解底物HY5以及BBX20–22、BBX24、PIF4和PIF5转录因子。同时, UVR8与转录因子WRKY36相互作用增强和稳定HY5的转录活性, HY5启动下游转录因子BBX11、BBX31和MYB12, 从而调控植物色素合成与光形态建成。UVR8可抑制MYB73/MYB77和BES1/BIM1的DNA结合活性, 也可促进MYB13和TCP4对下游基因的激活, 从而调控下游信号通路。实线箭头和实线T型分别表示激活和抑制; 虚线表示有待实验验证。

Figure 1 Model of transcription factors involved in plant's UV-B signaling pathway

Upon ultraviolet-B (UV-B) irradiation, its receptor UVR8 gets activated and subsequently depolymerizes into monomers, which then are translocated into the nucleus, instigating the downstream signaling pathways. UVR8 directly interacts with the E3 ubiquitin ligase COP1, resulting in the release of HY5, as well as the transcription factors BBX20–22, 24, PIF4 and PIF5 that would otherwise be degraded by COP1. On the other hand, UVR8 interacts with the transcription factor WRKY36 to potentiate and stabilize the transcriptional activity of HY5. HY5 triggers the downstream transcription factors BBX11, BBX31 and MYB12, orchestrating the regulation of plant pigment biosynthesis and photomorphogenesis. Moreover, UVR8 can inhibit the DNA-binding activities of MYB73/MYB77 and BES1/BIM1, and also promote the activation of downstream gene initiation by MYB13 and TCP4, thereby regulating the downstream signaling pathways. Solid arrows and T-lines represent activation and repression, respectively; the dotted lines represent that to be verified.

光照下稳定，并促进光形态建成(Zhang et al., 2017; Zeng et al., 2022)。HY5在UV-B辐照下稳定，可促进其对下游靶基因的诱导，抑制下胚轴伸长；而PIF4的活性在UV-B辐照下被UVR8抑制，导致下胚轴缩短，因此HY5与PIF4在参与UVR8介导的下胚轴伸长过程中具有拮抗作用(Küpers et al., 2020; Zhao et al., 2023; Sharma et al., 2023)。同时，UV-B能稳定COP1的靶基因*HFR1* (*LONG HYPOCOTYL IN FAR-RED 1*)，COP1通过与PIF4结合抑制其对下游靶基因的转录激活活性，从而抑制植物的光形态建成(Tavridou et al., 2020)。PIF5通过与COP1相互作用增强自身的稳定性，促进下胚轴的伸长和生长素的生物合成，而UVR8通过泛素-蛋白酶体系减弱PIF5与COP1之间的相互作用，加速PIF5的降解，抑制其调控靶基因表达的活性，从而抑制下胚轴的伸长(Sharma et al., 2019; Podolec et al., 2021)。

3 研究展望

光作为重要的环境因子，为植物生长发育所必需。光感受器在植物响应各种光照过程中发挥关键作用，转录因子在光信号通路中的作用同样重要。UV-B作为影响植物生长发育的环境因子，受到越来越多的关注，其中转录因子在调控网络中发挥核心作用(图1)。锌指转录因子BBX家族中除BBX11、BBX20–22、BBX24和BBX32外，还有其它成员参与调控UV-B响应，HY5转录因子在植物色素合成和光形态建成中发挥核心作用。*HY5*既受到BBX家族基因的调控，也可激活*BBX*家族基因的表达。深入研究*BBX*家族基因与*HY5*之间的调控网络，明确它们在UV-B信号通路中的作用机制和相互关系将有助于解析植物响应UV-B辐照的分子机制。植物在受到UV-B胁迫后，其表现是多面的，DNA损伤是其中较明显的特征，但在UV-B信号通路中DNA损伤的修复机制尚不明确。此外，UV-B辐射产生的作用伴随着剂量依赖效应，高剂量的UV-B辐射(常发生在高海拔地区)抑制植物生长甚至导致其死亡，被定义为一种环境胁迫；而低剂量的UV-B辐射作为信号调节植物生长(Mosadegh et al., 2019; Crestani et al., 2023; Wu et al., 2023)。植物在响应UV-B辐照时表现出的剂量依赖效应是如何形成的，UVR8是否在其中发挥关键作用，以及是否依赖转录因子的调控，相关机制尚不清楚。关于转录因子在UV-B信号通路中发挥作用的分子机制已有大量研究，但仍有很多问题需深入探索。UVR8在接受UV-B信号后，以单体形式在细胞核内结合COP1和相关转录因子，从而调控植物响应UV-B的信号通路。值得注意的是，这些转录因子之间是否存在相互联系或相互制约，在调控网络中这些转录因子相对独立还是相互作用，都是植物精确响应UV-B的关键所在。这些转录因子还可能参与对其它环境信号通路的调控，不同信号通路之间的交叉调控机制，以及它们如何整合多种环境信号协调植物的生长发育和抗逆反应将是未来的研究目标。

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The Regulatory Roles of the Transcription Factors in Plant's Response to UV-B Radiation

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Abstract UV-B, as an inherent constituent of sunlight, exerts a crucial influence on the growth and development of plants. Recent studies showed that UV-B is not merely an environmental stressor, but also a functional signal molecule that is able to promote plant growth under moderate radiation level. The protein UVR8, as a unique photoreceptor specific to UV-B, plays an irreplaceable role in the plant's response to UV-B, whose functions are regulated by both upstream and downstream transcription factors. At present, a variety of transcription factors such as BBXs, WRKYs, MYBs, and PIFs have been reported to be involved in UV-B regulated processes like hypocotyl elongation, primary root length, leaf size and shape, flowering cycle, and anthocyanin synthesis. This article reviews the molecular mechanisms of UVR8 in the UV-B signaling pathway and summarizes the regulatory mechanisms of the transcription factors during the UV-B radiation process, to provide reference for relevant research.

Key words UVR8, transcription factor, UV-B radiation, plant

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