综 述 Reviews

拟南芥下胚轴伸长与向光性的分子调控机理

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摘要: 下胚轴快速伸长和向光性是高等植物进行固着生活的重要适应性机制, 是子叶钻出土层进行光形态发生和光合作用的必要前提。拟南芥下胚轴因其简单的生理形态结构和特异的生理功能而成为剖析植物细胞伸长和向性生长的理想模式系统。本文主要介绍光和植物激素调控拟南芥下胚轴伸长和向光性弯曲的生理基础、遗传学功能及其分子调控机理的最新进展。

关键词: 拟南芥; 下胚轴; 细胞伸长; 向光性; PIFs; 激素

Molecular Regulatory Mechanisms of Hypocotyl Elongation and Phototropism in *Arabidopsis*

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Abstract: Rapid elongation and phototropism of the hypocotyl, which are essential for the young seedling to perceive light and undergo photomorphogenesis and photosynthesis, are important adaptive mechanisms for plant sessile life. The *Arabidopsis* hypocotyl is an ideal model system to dissect cell elongation and tropic growth in plants due to its simple physiological and morphological structure and specific physiological roles. This review focused on physiological basis and genetic functions of light- and phytohormone-regulated hypocotyl elongation and phototropism, and recent progresses on their regulatory molecular mechanisms in *Arabidopsis thaliana*.

Key words: *Arabidopsis thaliana*; hypocotyl; cell elongation; phototropism; PIFs; hormones

高等植物种子在土层中萌发后, 下胚轴(hypocotyl)迅速向上伸长, 将子叶推出土层, 并转向阳光一侧, 开始光合作用, 为幼苗植株后续生长发育提供充足的养分。下胚轴的快速向上伸长和向光性生长是高等植物长期进化的结果。如果下胚轴失去这些生长特性, 萌发的种子会因子叶或胚乳中的养分耗尽而死于土层中, 无法完成生活周期。因此, 下胚轴对高等植物尤其是双子叶植物的生命延续具有特殊的生理意义。下胚轴独特的生长特性引起了植物学家浓厚的兴趣, 具有重要的科学研究价值。大量的实验证据表明, 高等植物下胚轴是研究植物细胞伸长(cell elongation)和向光性(phototropism)分子机理的经典材料(Vandenbussche等2005)。最近几年, 植物学家利用遗传、细胞和生理生化等现代分析手段, 从分子细胞水平上剖析了下胚轴伸长和向光性的分子机理, 取得了长足的进展(Nozue等2007; de Lucas等2008; Feng等2008; Ding等2011; Sun等2013)。因此, 本文在介绍一些光和激素调控下胚轴的研究背景的基础上, 重点介绍最近几年有关下胚轴伸长和向光性生长的最新进展。

1 下胚轴的生理与形态结构基础

下胚轴是高等植物胚胎和胚后期的幼苗茎, 是连接根系和茎叶的重要结构, 也是水、矿质元素、养分和信号分子运输的重要通道; 同时, 对内源信号分子(如激素)和环境刺激(如光、温度和重力等)非常敏感, 并作出快速响应(Nozue等2007; de Lucas等2008; Feng等2008; Christie等2011; Ding等

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2011; Franklin等2011; Bai等2012; Sun等2012, 2013; Kami等2014)。因此, 下胚轴是一个可塑性很强的器官, 对高等植物幼苗的生长发育具有重要生物学功能。双子叶模式植物拟南芥(*Arabidopsis thaliana*)下胚轴形态结构简单, 从顶端到基部(纵向)共由20多个细胞组成, 大多在胚胎期形成, 仅少数在种子萌发后通过细胞分裂产生, 因此下胚轴伸长主要起因于细胞伸长。拟南芥下胚轴从内到外(横向)由中柱维管束、内皮层、皮层和表皮层等组成(Gendreau等1997)。拟南芥下胚轴因具有简单的形态结构和特异的生理功能而成为研究植物细胞伸长和向性生长的重要模式系统。

2 下胚轴伸长的调控机理

已知下胚轴的快速伸长受环境因子和多种内源激素的综合调控。最近的遗传与生化证据表明, 环境信号和内源激素通过转录因子光敏色素互作蛋白PIFs (PHYTOCHROME-INTERACTING FACTORs)整合调控下胚轴伸长(Nozue等2007; de Lucas和Prat 2014; Leivar和Monte 2014)。

2.1 光和激素调控下胚轴伸长的遗传学证据

在土壤中, 黑暗诱导萌发的种子下胚轴快速伸长, 导致子叶破土而出接受阳光, 标志着暗形态发生(skotomorphogenesis)的终止和光形态发生(photomorphogenesis)的开始。光抑制下胚轴伸长主要依赖于五类光受体: 光敏色素PHYA-E (PHYTOCHROMEs, 远红光或红光受体)、向光素PHOT (PHOTOTROPINs, UV-A/蓝光受体)、隐花色素CRY (CRYPTOCHROMEs, UV-A/蓝光受体)、ZTL/FKF1/LKP2家族(ZEITLUPE/FLAVIN BINDING KELCH-REPEATF-BOX1/LOV KELCH PROTEIN2, UV-A/蓝光受体)和UVR8 (UV-B RESISTANCE 8, UV-B受体) (Yang等2000; Möglich等2010; Wu等2012)。遗传分析表明, 突变体*phyA*和*phyB*分别在远红光和红光下具有长胚轴表型(Reed等1998), 表明PHYA/B介导远红光或红光对下胚轴伸长的抑制效应。在特定的UV-B照射下, 突变体*uvr8*具有长胚轴表型(Favory等2009), 表明UVR8介导UV-B对下胚轴伸长的抑制作用。在蓝光下, 突变体*phot1*下胚轴长度与野生型相似(Kang等2008), 而*cry1*下胚轴明显比野生型长(Parks等1998), 表明CRY1介导蓝光对下胚轴伸长的抑制作用。然而, 蓝光能瞬时抑制黑暗生长的野生型、*phot2*和*cry1*下胚轴伸长, 但未能瞬时抑制*phot1*和*phot1cry1*下胚轴伸长; 相反, 蓝光能持续抑制野生型和*phot1*, 但不能持续抑制*cry1* (Folta等2001), 暗示PHOT1介导蓝光对下胚轴的快速抑制。*ztl-1*突变体下胚轴在蓝光下比野生型长3倍, 但在红光下其伸长受到抑制(Somers等2000), 表明ZTL介导蓝光对下胚轴伸长的调控作用。

植物激素是下胚轴伸长的重要调控信号分子。已知外源生长素、赤霉素(GA)和油菜素甾醇(BR)处理促进下胚轴伸长(Vandenbussche等2005), 暗示这些生长物质正调控下胚轴伸长。生长素转录信号突变体*axr1-12* (*auxin resistant 1-12*) (Leyser等1993)和核受体突变体 *tir1afb2afb3* (*transport inhibitor response 1/auxin signaling f-box protein 2/3*)均表现为短胚轴, 并且对外源生长素处理不敏感(Chapman等2012), 表明生长素转录信号途径对下胚轴伸长是必需的。GA合成突变体*ga1*和信号突变体*gai*具有短胚轴表型, 外源GA_4处理能恢复*ga1*短胚轴表型, 但未能恢复*gai*短胚轴表型(Cowling和Harberd 1999)。同样, BR合成突变体*dwf1/dim/cbb1* (*dwarf 1/dimunito/cabbage 1*)和*det2* (*de-etiolated 2*)和BR信号突变体*bri1* (*brassinosteroid-insensitive 1*)均表现为短胚轴表型(Clouse 1996)。这些分析结果表明, GA和BR合成与信号传导对下胚轴伸长也是必需的。

2.2 下胚轴伸长的分子调控机理

早期的遗传与生化证据表明, 拟南芥下胚轴伸长主要受外界光信号和内源激素拮抗调控, 但具体的拮抗调控机制一直不清楚。最近的研究发现, 转录因子PIFs通过整合胞内外信号来调控下胚轴伸长(图1)。PIFs是一类含bHLH (basic helix-loop-helix)结构域的转录因子家族, 最初的生物信息学分析暗示, 在PIF亚家族中, 共有15个同源成员(Toledo-Ortiz等2003), 但至今仅7个成员(PIF1、3、4、5、6、7和8)被证实能与PHYA或PHYB互作(Leivar和Monte 2014) (图1)。这7个PIFs蛋白主要由bHLH结构域(与DNA结合)、APB结构域(active PHYB binding; 与PHYB活性形式Pfr结合)和APA结构域(active PHYA binding, 与PHYA活性形式Pfr结合)组成(Leivar和Monte 2014)。

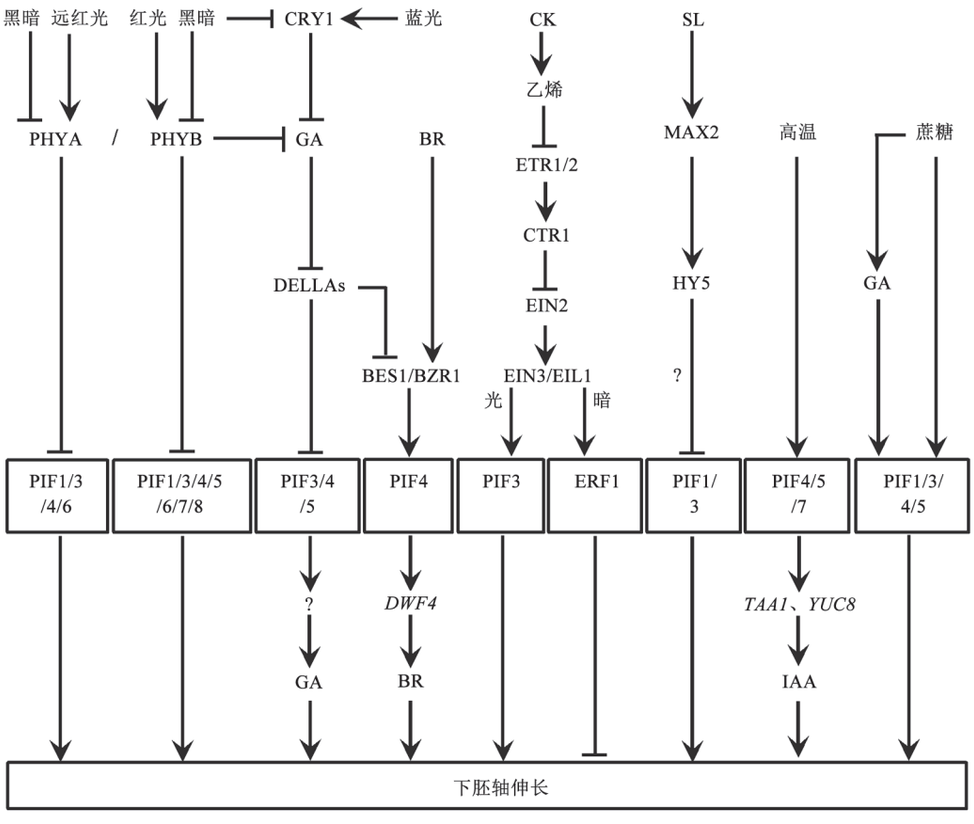

图1 下胚轴伸长的分子调控机理

Fig.1 Molecular regulatory mechanisms of hypocotyl elongation

参考de Lucas等(2008)、 Feng等(2008)、Bai等(2012)、Sun等(2012)、Leivar和Monte (2014)文献并作修改。

最近的研究表明, 黑暗生长的幼苗受到外界光信号刺激后, PHYA和PHYB分别被远红光和红光激活后以Pfr形式进入细胞核, PHYA与PIF1/3/4/6结合, 而PHYB与PIF1/3/4/5/6/7/8都能结合, 以诱导这些PIFs磷酸化和快速降解, 从而抑制由这些PIFs介导的暗形态发生和下胚轴伸长(Nozue等2007; Shen等2007; Leivar等2008; Shen等2008; Jeong等2013)。四突变体*pif1pif3pif4pif5* (*pifq*)在黑暗下表现出与组成型光形态发生突变体*cop1* (*constitutive photomorphogenesis 1*)相似的表型, 如短胚轴、子叶展开等(Leivar等2008), 同样, PIF1/3/4/5功能同时缺失抑制了*phyB*长胚轴表型(de Lucas等2008; Leivar等2012); 相反, 分别过表达PIF3、PIF4、PIF5导致长胚轴等暗形态发生表型(Khanna等2007; Sun等2012; Jeong等2013)。以上遗传与生化证据表明光通过激活PHYA/B的活性来促进PIFs降解从而抑制下胚轴伸长, 而黑暗通过抑制PHYA/B的活性来促进PIFs的积累和下胚轴伸长(图1)。

已知光信号能调控植物内源激素的生物合成。PHYA/B或CRY1功能缺失促进GA含量上升和下胚轴伸长(Reed等1998; Foo等2006; Kunihiro等2010)。GA促进受体GID1 (GA INSENSITIVE DWARF 1)与其底物转录阻碍蛋白DELLAs结合, 导致DELLAs泛素化和降解。最近的研究发现, PHYB和CRY1通过调控GA含量来调控DELLAs水平, 而DELLAs通过与PIF3/4/5互作从而抑制这些PIFs与DNA的结合能力和转录活性(de Lucas等2008; Feng等2008; Kunihiro等2010), 表明PIFs位于DELLAs下游起作用(图1), 暗示光通过下调GA含量促进DELLAs积累, 从而导致PIF3/4/5转录活性的下调和下胚轴伸长; 相反, 黑暗促进GA合成和DELLAs降解, 从而释放PIF3/4/5的转录活性和下

胚轴伸长。因此, 光信号通过PHYA/B-或CRY1-GA-DELLA途径来调控PIF3/4/5的转录活性和下胚轴伸长(图1), 推测*della*多突变体长胚轴表型可被PIF3/4/5功能同时缺失所抑制。

最近的生化分析表明, PIF4与调控BR信号传导的转录因子BES1/BZR1 (BRI1-EMS-SUPPRESSOR 1/BRASSINAZOLE RESISTANT 1)结合, 以异源二聚体(BZR1-PIF4)的形式激活下游靶基因的表达(Oh等2012; de Lucas和Prat 2014) (图1)。*pifq*下胚轴对外源BR不敏感, 并且抑制了功能获得性突变体*bzr1-D* (组成型非磷酸化活性形式)长胚轴表型(Oh等2012), 表明BES1/BZR1介导下胚轴伸长位于PIFs的上游。遗传与生化证据表明, DELLAs通过同时与BZR1和PIF4互作从而抑制BZR1-PIF4二聚体的转录活性; 相反, GA释放DELLA对BZR1的抑制效应(图1); *pifq bzr1-1D*突变体对GA不敏感(Bai等2012), 表明激活的BZR1对GA诱导下胚轴伸长是必需的, 并且DELLA-BZR1-PIF4之间的互作是光信号、GA和BR调控下胚轴伸长的重要机制之一(图1)。另有研究表明, BZR1结合于*IAA19*和*ARF7*启动子(Zhou等2013), 而PIF4与*IAA19*和*IAA29*启动子结合(Sun等2013), 暗示BZR1-PIF4对生长素下游基因的转录调控是BR与生长素交互对话(crosstalk)的重要机理之一。

另外, 其他激素如乙烯、细胞分裂素(CK)和独脚金内酯(SL)抑制黑暗诱导的下胚轴伸长(Vandenbussche等2005; Jia等2014)。乙烯在光下能促进下胚轴伸长, 但在黑暗下却抑制下胚轴伸长(Smalle等1997)。在黑暗中, 乙烯过量合成突变体*eto1* (*ethylene overproducer 1*)、信号传导突变体*ctr1* (*constitutive triple response 1*)和受体三突变体*etr1etr2ein4* (*ethylene response 1*/*2*/*ethylene insensitive 4*)均表现为短胚轴(Guzman和Ecker 1990; Hua和Meyerowitz 1998), 而乙烯信号传导突变体*ein2*具有长胚轴表型, 对乙烯不敏感(Cary等1995)。因此, 乙烯受体ETR1激活CTR1从而抑制下游EIN2和EIN3/EIL1 (ETHYLENE INSENSITIVE 3/EIN3 like 1)介导的乙烯信号途径(Guo等2003)。最新研究表明, 光通过促进PIF3降解和提高转录因子ERF1 (ETHYLENE RESPONSE FACTOR 1, 负调控下胚轴伸长) 稳定性来抑制下胚轴伸长(Zhong等2014)。在光下, 乙烯通过促进EIN3/EIL1介导的PIF3转录活性从而促进下胚轴伸长; 而在黑暗下, 乙烯通过提高EIN3/EIL1介导的ERF1稳定性从而抑制下胚轴伸长(Zhong等2012, 2014) (图1), 暗示在光和黑暗下乙烯对下胚轴伸长的截然相反的调控作用主要通过调控EIN3/EIL1介导的PIF3转录活性和ERF1稳定性来实现的。细胞分裂素CK在黑暗下抑制下胚轴伸长但在光下不影响下胚轴伸长。突变体*ckr1* (*cytokinin resistant 1*)/*ein2*和*ein1*下胚轴伸长对细胞分裂素BA (6-benzylaminopurine)和乙烯不敏感(Cary等1995), 表明细胞分裂素通过乙烯来间接影响下胚轴伸长(图1)。SL在黑暗下抑制下胚轴伸长(Cheng等2013)。光受体突变体*cry1*、*cry2*、*phyA*和*phyB*及其下游*cop1*和 *pifq*在相应的单色光下对独脚金内酯类似物GR24超敏(Jia等2014), 暗示CRY和PHYA/B介导的光信号途径负调控SL对下胚轴伸长的影响。GR24 (<10 μmol·L^{-1})能有效抑制光下野生型下胚轴伸长, 而SL信号突变体*max2* (*more axillary growth 2*)下胚轴伸长对GR24不敏感(Jia等2014), 暗示SL通过MAX2介导的信号途径负调控下胚轴伸长。另外, SL通过促进MAX2介导的bZIP转录因子HY5 (ELONGATED HYPOCOTYL 5)的转录活性和光依赖的HY5积累来抑制下胚轴伸长(Jia等2014) (图1)。尽管已知PIF1或PIF3能与HY5形成异源二聚体(Chen等2013), 但这种异源二聚体在下胚轴伸长中的作用仍有待于进一步证实(图1)。

已知高温(29 ℃)促进下胚轴局部生长素合成从而诱导下胚轴伸长(Gray等1998)。最近的遗传与生化证据表明, PIF4、PIF5和PIF7能与生长素合成酶基因*TAA1* (*TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS 1*)和*YUC8* (*YUCCA 8*)启动子结合从而来激活*TAA1*和*YUC8*的表达(Franklin等2011; Hornitschek等2012; Sun等2012)。YUC8功能缺失显著抑制由*PIF4*过表达或高温诱导的长胚轴表型(Sun等2012), 表明高温通过促进PIF4的表达从而激活*YUC8*的转录, 最终导致下胚轴IAA含量上升和下胚轴伸长(图1)。除促进下胚轴IAA合成外, 高温也间接诱导BR合成酶基因*DWF4* (*DWARF4*)的表达和BR合成, 从而促进了BR介导的下胚轴伸长(Maharjan和Choe 2011) (图1)。另外,

外源蔗糖能诱导野生型下胚轴伸长, 但未能诱导*pifq*下胚轴伸长且外源GA未能弥补这一缺陷(Liu等2011), 暗示PIF1/3/4/5和GA共同介导蔗糖诱导的下胚轴伸长(图1)。

综上所述, 虽然PIF家族中的成员对下胚轴伸长都有一定的作用, 但不同PIFs在不同环境或激素信号传导中起作用, 它们之间的功能并不是完全冗余。在黑暗中, PIF1参与抑制种子萌发、下胚轴顶钩的形成和子叶的打开; 而在光下, PIF3参与调控乙烯诱导的下胚轴伸长(Jeong等2013)。PIF4介导高温诱导下胚轴伸长, 而PIF7介导下胚轴避荫反应(Li等2012); PIF3/4/5与DELLA互作, 而PIF4/5/7调控生长素的合成。

3 下胚轴向光性调控机理

植物向光性是植物根据光源方向通过下胚轴或茎的向光面和背光面的差异生长来改变自身的生长方向(Sakai和Haga 2012)。这是植物为了争夺有限光源和生存空间, 在进化过程中逐渐形成的一种环境适应性机制。已知单侧蓝光或白光诱导的下胚轴向光性弯曲主要由质膜定位的PHOT来介导, 由三个关键性步骤组成: (1) PHOT感受并传导蓝光信号, (2)下胚轴向光面与背光面生长素不对称分布, (3)下胚轴细胞不对称伸长和向光性弯曲(Hohm等2013)。在此过程中, 其他光信号受体和激素也参与调控下胚轴向光性弯曲。

3.1 PHOT介导蓝光诱导的向光性

拟南芥蓝光受体PHOT家族共有两个同源成员PHOT1和PHOT2。遗传分析表明, 在单侧弱蓝光下, 突变体*phot1*和*phot1phot2*下胚轴均无向光性表型, 但*phot2*仍有向光性; 在单侧强蓝光下, *phot1phot2*仍无向光性, 但*phot1*和*phot2*均有向光性(Sakai等2001), 表明PHOT1和PHOT2之间存在部分功能冗余, PHOT1是强弱蓝光诱导向光性弯曲所必需的, 而PHOT2仅在强蓝光下起作用。

PHOT主要由N端的光感受结构域和C端的Ser/Thr激酶域组成。Ser/Thr激酶域主要催化蓝光激发的PHOT自体磷酸化(Cho等2007), 而N端含有LOV1 (light oxygen voltage 1)和LOV2光感受结构域(Demarsy和Fankhauser 2009), 其中LOV2是PHOT1自体磷酸化和PHOT1介导下胚轴向光性不可或缺的(Cho等2007), 但LOV1的功能目前仍不清楚。在LOV2和激酶区域之间的活性环中, PHOT1 Ser851残基非常保守, 它的自体磷酸化是光信号转导的主要分子事件(Inoue等2008)。Ser/Thr蛋白磷酸酶2A (PP2A)功能缺失引起PHOT2去磷酸化抑制和向光性弯曲的加强(Tseng和Briggs 2010), 表明PP2A对PHOT2介导的向光性具有负调控作用。

质膜定位的PHOT1通过网格蛋白介导的内吞CME (clathrin-mediated endocytosis)途径进入胞腔内, 蓝光促进PHOT1内吞(Kaiserli等2009), 而这一过程被红光诱导的PHYA活性所抑制, 从而促进向光性弯曲(Han等2008)。此外, PHOT1与生长素运输载体PIN (PIN FORMED)和ABCB (ATP-BINDING CASSETTE B)蛋白共定位于质膜(Wan等2008; Titapiwatanakun等2009), 暗示PHOT1可能与PIN和ABCB之间存在一定的互作关系。目前已知拟南芥有四种PHOT互作蛋白: NPH3 (NON-PHOTOTROPIC HYPOCOTYL 3)、RPT2 (ROOT PHOTOTROPISM 2)、PKS1 (PHYTOCHROME KINASE SUBSTRATE 1)和14-3-3 (Chen等2008; Hohm等2013)。

NPH3和RPT2属于NRL (NPH3/RPT2-like)家族成员, 拥有BTB/POZ (BROAD COMPLEX, TRAMTRACK, BRIC A BRAC/POX VIRUS AND ZINC FINGER)和coiled-coil结构域, 参与蛋白与蛋白之间的互作(Motchoulski和Liscum 1999)。质膜定位的NPH3与PHOT1/2互作(Sakamoto和Briggs 2002)。蓝光分别诱导NPH3去磷酸化和PHOT1磷酸化(Pedmale和Liscum 2007), NPH3参与调控PIN的亚细胞定位(Wan等2012), NPH3功能缺失抑制了下胚轴生长素的不对称分布和向光性弯曲(Haga等2005; Roberts等2011), 表明NPH3去磷酸化对于PHOT1介导的向光性具有重要调控作用。然而, 强蓝光诱导的*phot1*单突变体的向光性应答中, NPH3并没有去磷酸化(Tsuchida-Mayama等2010), 暗示PHOT2介导的向光性不需要NPH3去磷酸化。同样, 质膜定位的RPT2与PHOT1共定位(Inada等2004)。酵母双杂交分析表明, RPT2与NPH3互作(Lariguet等2006)。RPT2参与调控PHOT1介导的向光性弯曲, 尤其在强蓝光下(Sakai等2000; Inada等2004; Sakai和Haga 2012), 但并不参与PHOT2介导的向光性弯曲(Zhao等2013)。这些分

析结果暗示PHOT1和PHOT2通过不同调控机制来介导下胚轴向光性。PKS也是质膜定位蛋白, 共有4个同源成员PKS1~PKS4 (Lariguet等2006), 其中PKS4主要在下胚轴伸长区表达, 体外磷酸化分析暗示PKS4很可能是PHOT1的磷酸化底物(Demarsy等2012)。*pks1*、*pks2*和*pks4*的单和双突变体均有向光性缺陷表型, 而*pks1pks2pks4*向光性缺陷更加严重(Lariguet等2006), 且在该突变体的黄化幼苗顶钩区域, 生长素侧向运输及其信号传导发生改变(Kami等2014), 表明PKS之间存在功能冗余。有研究表明PKS1均能与PHOT1/2和PIN1相互作用(Lariguet等2006; Zhao等2013), 暗示PHOT1/2可能通过PKS1来调控PIN1的亚细胞定位(图2)。另外, 14-3-3λ主要与PHOT介导的气孔开启相关, 是否参与调控下胚轴向光性仍缺乏证据。以上结果表明, NPH3、RPT2和PKS是PHOT介导向光性的重要互作蛋白。

除PHOT外, 其他光受体也参与向光性弯曲。在蓝光处理之前, 用UV-A、红光、远红光预处理或和蓝光一起处理能够增强下胚轴向光性弯曲(Liscum和Briggs 1996)。遗传分析表明, PHYA功能缺失抑制了红光对向光性的促进作用, 但PHYB功能缺失并没有影响红光的增强作用(Parks等1996)。最近的分析表明, *phyAphyBphyCphyDphyE*五突变体仍有90%的向光性反应, 但*phyBphyCphyDphyE*四突变体仍有正常向光性(Kami等2012), 表明向光性的增强效应主要由PHYA来介导。更有意思的是, *phyAcry1cry2*和*phyAphyBcry1cry2*具有严重的向光性缺陷(Tsuchida等2010), 表明PHYA和CRY具有协同调控向光性的功能(图2)。

3.2 生长素介导的向光性弯曲

生长素不对称分布是导致向性生长的主要原因, 这种不对称分布主要由生长素运输载体AUX1/LAX (AUXIN RESISTANT 1/LIKE AUXIN RESISTANT)、ABCB19和PIN协同完成(Christie等2011; Ding等2011; Zhang等2013)。最近的遗传与生理分析表明, 单侧蓝光或白光诱导野生型下胚轴伸长区PIN3的重定位, 引起内皮层细胞背光一侧有更多的PIN3定位(侧向定位), 导致下胚轴生长素的不对称分布和向光性弯曲; 相反, PHOT1功能缺失抑制了单侧光诱导的下胚轴PIN3重定位、生长素不对称分布和向光性弯曲, 同样, *pin3*下胚轴生长素不对称分布和向光性弯曲也均被抑制(Ding等2011), 而且 *pin3pin4pin7*和*pin1pin3pin7*下胚轴向光性缺陷比*pin3pin7*更严重(Haga和Sakai 2012)。此外, 在Ser/Thr蛋白激酶*PID* (*PINOID*)过表达突变体和*wag1wag2pid*缺失突变体中, 单侧光诱导的PIN3侧

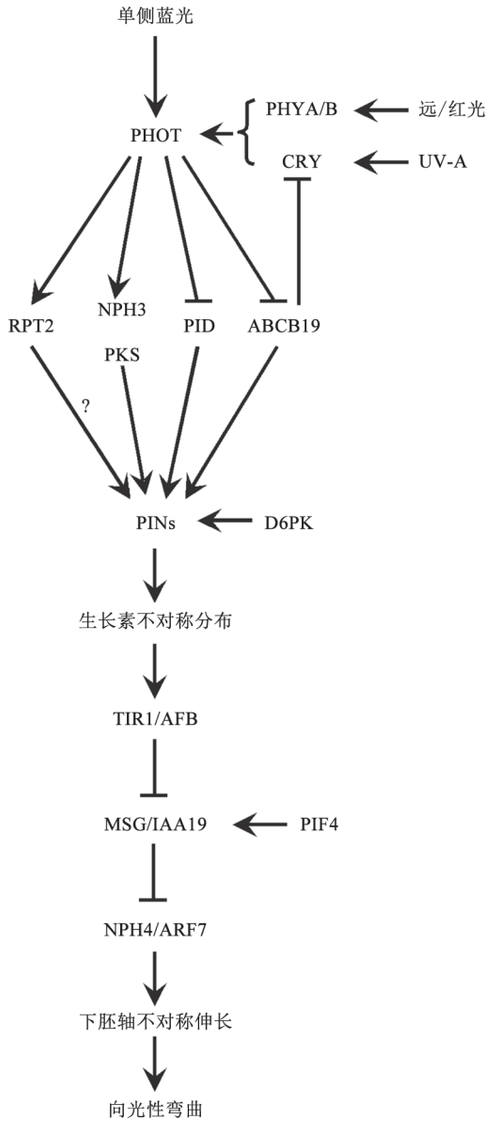

图2 下胚轴向光性分子调控机理

Fig.2 Molecular regulatory mechanisms of hypocotyl phototropism

参考Zourelidou等(2009)、Christie等(2011)、Ding等(2011)、Sun等(2013)文献并作修改。

向定位和向光性弯曲均被抑制; 进一步分析表明, 蓝光显著抑制野生型植株*PID*的转录水平, 而在*phot1phot2*双突变体中这种抑制作用明显减弱(Ding等2011), 暗示PHOT介导的蓝光信号通过抑制*PID*的转录水平和PIN3磷酸化来促进PIN3侧向重定位, 从而诱导生长素不对称分布和向光性弯曲(图2)。然而, 另有研究认为, 单侧蓝光诱导下胚轴生长素不对称分布最初发生在下胚轴顶部(子叶节) (Christie等2011)。在下胚轴顶部, 蓝光促进PHOT1介导的ABCB19磷酸化从而抑制ABCB19的输出活性, 引起下胚轴垂直生长停止和顶部背光面生长素积累, 生长素由此侧向流向下胚轴伸长区背光面, 最终导致向光性弯曲(Christie等2011)。ABCB19功能缺失促进向光性弯曲, 可能通过激活PHY和CRY的功能来实现(Noh等2003; Nagashima等2008; Christie等2011)。这些结果暗示ABCB19负调控下胚轴向光性弯曲(图2)。此外, 生长素输入载体AUX1/LAX功能缺失后也引起下胚轴向光性缺陷, 表明AUX1/LAX参与调控下胚轴向光性(Stone等2008), 但具体的调控机理仍不清楚。

除生长素运输载体外, D6PK (D6 PROTEIN KINASE)和生长素信号传导途径也参与调控下胚轴向光性弯曲。遗传与生化分析表明, D6PK能磷酸化PIN3蛋白, D6PK功能缺失多突变体*d6pk012*和*d6pk0123*下胚轴生长素向基运输和向光性弯曲均被显著抑制(Willige等2013), 表明D6PK通过介导PIN磷酸化调控下胚轴生长素运输和向光性弯曲(图2)。阻碍蛋白MSG2/IAA19 (MASSUGU 2/INDOLE-3-ACETIC ACID 19)通过抑制转录因子NPH4/ARF7 (AUXIN RESPONSE FACTOR 7)的活性, 从而参与调控下胚轴向光性弯曲(Zourelidou等2009)。NPH4/ARF7功能缺失突变体(Harper等2000)和MSG2/IAA19功能获得性突变体(Tatematsu等2004)下胚轴向光性弯曲均被抑制。在单侧光刺激下, 下胚轴背光面的生长素大量积累促进了MSG2/IAA19降解, 释放NPH4/ARF7的转录活性(Stone等2008), 最终导致与向光性生长相关基因的表达(图2)。最近的遗传与生化证据表明, PIF4通过与*IAA19*和*IAA29*启动子G-box基序结合直接激活它们的表达, 从而抑制ARF7的活性和向光性弯曲; PIF4功能缺失促进了下胚轴向光性弯曲(Sun等2013)。TIR1/AFB介导的信号途径通过调控ARF7或其他转录因子的表达和生长素不对称分布, 从而引起向光性弯曲(Sakai和Haga 2012)。*tir1afb1afb2afb3*四突变体下胚轴向光性严重缺陷(Möller等2010)。因此, 推测生长素通过TIR1/AFB下调IAA19水平从而促进ARF7的表达和向光性弯曲, 相反, PIF4通过促进*IAA19*的表达来抑制ARF7的转录活性和向光性弯曲(图2)。

4 展望

近几年来, 人们综合利用遗传学、细胞学和生理生化等研究手段, 对植物下胚轴伸长和向光性机制研究已取得了重要进展, 如光信号和内源生长激素通过PIFs的整合调控作用来介导下胚轴伸长, ABCB和PIN介导下胚轴向光性弯曲的分子作用机理等等, 但仍有许多问题有待于进一步研究: (1)外界环境因子是否都通过PIF的整合功能来调控下胚轴伸长? (2)除PHYA/B外, PIFs是否与其他光受体如PHOT、CRY等互作从而调控蓝光信号途径? (3) PHOT互作蛋白如NPH3、RPT2和PKS等是否调控ABCB19的活性或PIN的重定位? (4)生长素胞外受体ABP1 (AUXIN BINDING PROTEIN 1)和网格蛋白是否参与调控下胚轴伸长和向光性? 如何调控? 因此, 要真正剖析植物下胚轴伸长和向光性分子机理仍面临巨大的挑战。

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