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·专题论坛·

光敏色素信号通路中磷酸化修饰研究进展

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摘要 光是植物的唯一能量来源, 植物在进化过程中产生不同的光敏色素来感知光信号。光信号通路中元件通常被特异翻译后修饰调节。光敏色素是一种自磷酸化的丝氨酸/苏氨酸蛋白激酶, 可以被一些蛋白磷酸酶去磷酸化。通过对光敏色素A (phyA)和光敏色素B (phyB)的自磷酸化位点研究, 发现自磷酸化对光敏色素的功能及其介导的信号通路起着非常重要的作用。光激活的光敏色素诱导光敏色素作用因子(PIF)磷酸化, 这对于PIF的正常降解及光形态建成的起始是必需的。该文主要介绍了光敏色素信号通路磷酸化修饰的最新进展, 以期为深入研究光敏色素信号转导机制提供参考。

关键词 光敏色素, 光敏色素作用因子, 磷酸化修饰

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植物种子萌发后, 在生长发育过程中需要适应多种环境因子, 包括温度、光、湿度和盐度等。在这些因子中, 光最为重要。植物依赖光受体接收不同的光, 光敏色素(phytochrome, phy)作为红光/远红光受体接受光信号后, 将其传递到核内光响应因子, 最终调控植物的光形态建成。在光信号通路中, 磷酸化和去磷酸化作为一种重要的翻译后修饰被广泛研究。本文基于前人的研究成果, 对光敏色素信号通路的磷酸化修饰进行了综述和讨论。

1 光敏色素调控植物发育

光敏色素作为一类光受体, 以红光/远红光(R/FR)依赖的形式存在。在模式植物拟南芥(*Arabidopsis thaliana*)中, 光敏色素调控3 000多个基因的表达, 并且在植物适应光环境的急剧变化中起非常重要的作用(Chen et al., 2004)。光敏色素是植物体内合成的一种调节生长发育的蛋白, 含2个主要结构域: N端感光区和信号区及C端二聚化区和定位区(又称光调节区) (Rockwell et al., 2006; Fankhauser and Chen, 2008; Nagatani, 2010; Ulijasz et al., 2010)。光感受区和光调节区通过铰链区连接在一起(王静和王艇, 2007)。phys以2种相对稳定的形式存在: 红光吸收无活性形式Pr和远红光吸收激活形式Pfr。有活性的光敏色素A (phyA)和光敏色素B (phyB)可以与一些特异的细胞分子相互作用, 这对于它们转运到细胞核内是必需的(Hiltbrunner et al., 2005; Rockwell et al., 2006; Pfeiffer et al., 2012)。在此互作过程中光敏色素信号被级联传递(Bae and Choi, 2008), 表明有活性的光敏色素分子与细胞分子间的相互作用程度决定了光敏色素信号的强弱。在拟南芥中, phys家族包括5个成员, 即从phyA到phyE, 它们可以相互形成同源和异源二聚体(Clack et al., 2009)。phyA和phyB是最主要的光敏色素, 参与调节植物生长发育的各个方面(Franklin and Quail, 2010; Kami et al., 2010)。phyA作为一种远红光感受器, 调节从暗形态建成到光形态建成的转变。phyB作为红光调节分子开关, 在红光信号通路中起着非常关键的作用(Nagy and Schäfer, 2002)。光敏色素不同的作用模式为黄化幼苗在远红光或者红光下产生不同的形态学响应提供了一定的分子基础。

在拟南芥中, 发现了许多与光敏色素信号分子相关的突变体(Chory, 2010)。这些光形态突变体可以分成2大类。第1类突变体表现去黄化和持续光形态表型, 包括下胚轴短、子叶膨大和部分叶绿体分化, 以及在黑暗条件下光诱导基因的表达去抑制等。这些突变体包括*de-etiolated 1* (*det1*) (Chory et al., 1989)、*constitutively photomorphogenic 1* (*cop1*) (Deng et

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al., 1991)、*COP9*的信号组分突变体(Serino and Deng, 2003)、*suppressor of phytochrome A-1051-4* (*spa1-4*) (Laubinger et al., 2004)和*phytochrome interacting factors 1,3,4,5* (*pifQ*) (Leivar et al., 2008b, 2009; Shin et al., 2009)。鉴于它们的基因突变都是隐性的, 被定义为光信号通路中的负向调节组分。此外, 这些突变体的表型与组成型激活*phyB*等位基因的突变体*YHB* (*Y*^{276}*H mutant of phyB*)非常相似(Su and Lagarias, 2007; Hu et al., 2009), 表明phys通过抑制这些负调节子来启动光形态建成。

第2类突变体表型都与*phyA*和*phyB*功能缺失突变体类似, 在光下均表现出下胚轴变长和子叶紧闭表型, 这些突变体在phyA或phyB信号中存在缺陷, 或者在2个信号通路中都存在缺陷。例如: *far-red elongated hypocotyl 1* (*fhy1*) (Whitelam et al., 1993; Desnos et al., 2001)、*fhy3* (Whitelam et al., 1993; Wang and Deng, 2002)、*long after far-red light 1* (*laf1*) (Ballesteros et al., 2001)、*far-red impaired response 1* (*far1*) (Hudson et al., 1999)和*long hypocotyl in far-red 1* (*hfr1*) (Fairchild et al., 2000)等, 在phyA信号通路中存在缺陷, 而*the elongated hypocotyl 5* (*hy5*)突变体在phyA和phyB信号通路中都表现出缺陷(Koornneef et al., 1980; Oyama et al., 1997)。最近报道的*hemera* (*hmr*)是一种新的phy信号突变体, 它不仅在红光和远红光下表现出长的下胚轴, 而且在叶绿体发育方面也存在缺陷(Chen et al., 2010)。由于HMR在细胞核和叶绿体都有定位, 所以可能是一个双重功能蛋白。

上述2类突变体中许多基因编码光响应基因的转录调节子, 包括正向调节因子(HY5、LAF1、HFR1、FHY3和FAR1)和负向调节因子(PIFs)。光敏色素调节转录调节子的稳定性是调节基因表达的一个关键机制(Chen and Chory, 2011)。

2 光敏色素磷酸化修饰在光信号通路中的作用

2.1 phyA的磷酸化与去磷酸化作用

光敏色素是一类光调节的His激酶(Yeh et al., 1997)。自磷酸化的燕麦(*Avena sativa*) phyA在体外是由生色团调节的(Yeh and Lagarias, 1998)。通过研究纯化的光敏色素提取物, Hunt和Pratt (1980)发现光敏色素是一类磷蛋白。另有学者对燕麦幼苗进行了研究, 鉴定出光敏色素的3个磷酸化位点(Lapko et al., 1996, 1997, 1999)。其中, 2个磷酸化位点(Ser8和Ser18)位于phyA的N端延伸区(N-terminal extension, NTE); 另一个位点(Ser598)位于N端和C端之间的铰链区域。通过比对单子叶和双子叶植株中phyA的这3个磷酸化位点, 发现它们很保守(图1)。Cherry等(1992)报道NTE区域对于phyA的生物活性是必需的。将NTE区域的Ser8和Ser18位点替代为Ser8Ala和Ser18Ala可以抑制phyA的磷酸化作用, 从而增强phyA的生物活性。在转基因植物中表达这2个突变的phyA蛋白, 植株表现出对远红光敏感, 并且出现矮化表型(Stockhaus et al., 1992)。这些研究结果说明, NTE区域的磷酸化导致了phyA信号衰减, 是光敏色素信号转导的脱敏过程(Emmler et al., 1995; Jordan et al., 1996, 1997; Casal et al., 2002), 但是这些磷酸化位点的替代使得信号衰减的机制仍不清楚。Han等(2010)的研究发现模拟持续磷酸化的phyASer8-Asp和phyASer18Asp在植物中的降解速率变快。对水稻(*Oryza sativa*)中phyA的研究表明, 水稻phyA N端区域的Ser替换为Ala后转入烟草(*Nicotiana tabacum*), 同样可以导致转基因烟草对远红光非常敏感(Stockhaus et al., 1992)。该结果可以进一步解释为什么在拟南芥中将phyA的Ser8和Ser18位点模拟磷酸化, 可以减弱远红光调控的光响应, 而模拟去磷酸化的效应正好相反。

将燕麦phyA的另一个磷酸化位点Ser598替换为Ala的转基因植株*phyA*ser598Ala是一种有活性的特异性磷酸化位点突变体, 它对光表现出非常强的敏感性, 说明光敏色素Ser598位点磷酸化对于植物的光敏感性起抑制作用。有研究表明, phyA在体外被PKA (protein kinase A)催化发生磷酸化后, 其蛋白光谱和构象都没有发生变化(Lapko et al., 1996)。同时检测野生型燕麦phyA与phyASer598Ala中phyA的降解, 发现蛋白降解也没有变化, 说明Ser598Ala替换并不影响phyA的蛋白降解。在黑暗条件下燕麦phyA-GFP与phyAser598Ala-GFP没有表现出核定位, 在红光条件下, 2种phyA-GFP融合蛋白都表现出核定位, 并且形成核斑点, 这些核斑点之间没有定性和定量的变化。这些研究结果表明, Ser598Ala突变没有影响phyA

光诱导的入核以及核斑点的形成。虽然phyAser598Ala仍然保持自磷酸化能力，但是phyA突变为phyA-ser598Ala后会影响其与nucleoside-diphosphate kinases (NDPK2)的相互作用(Choi et al., 1999)，同

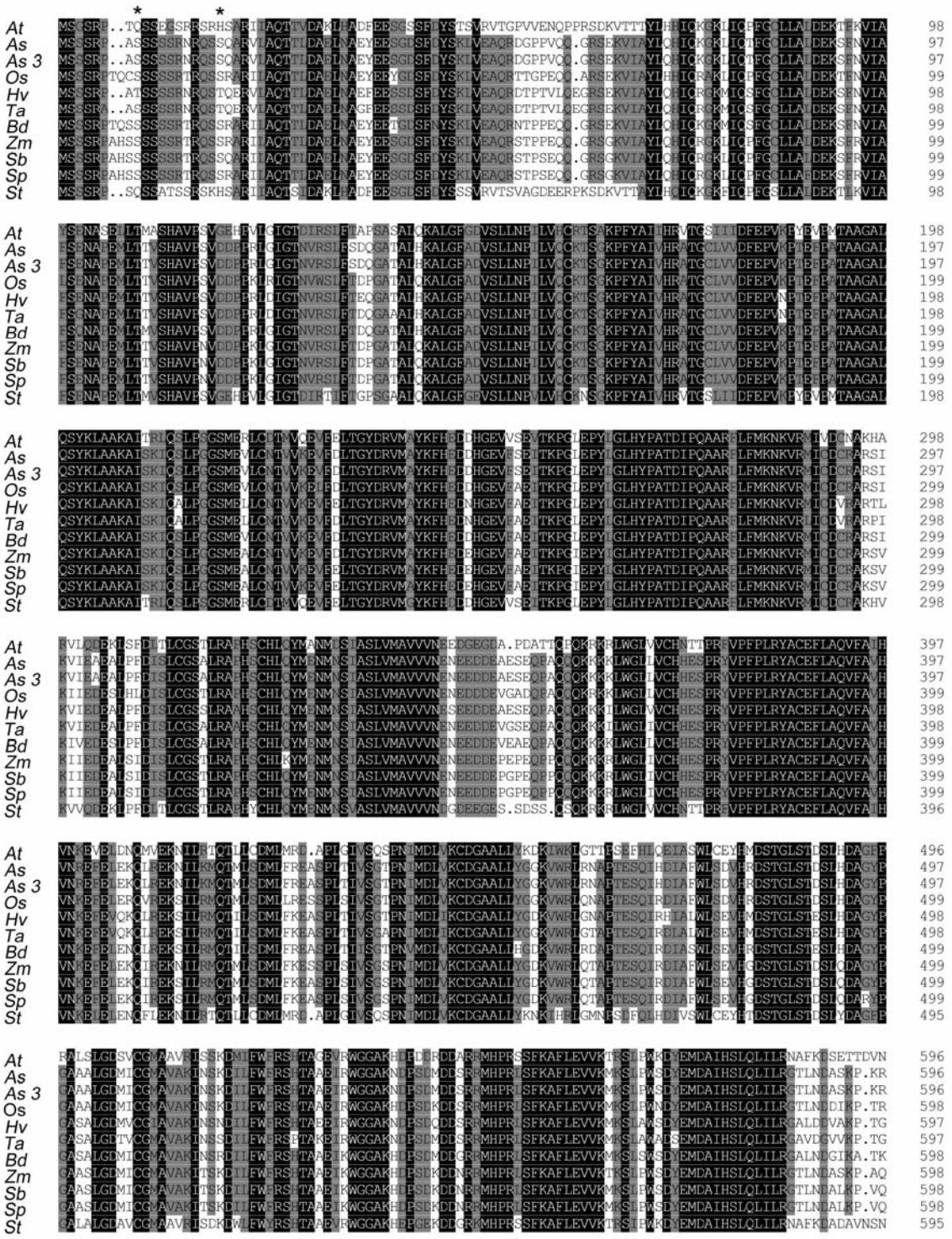

图1

Figure 1

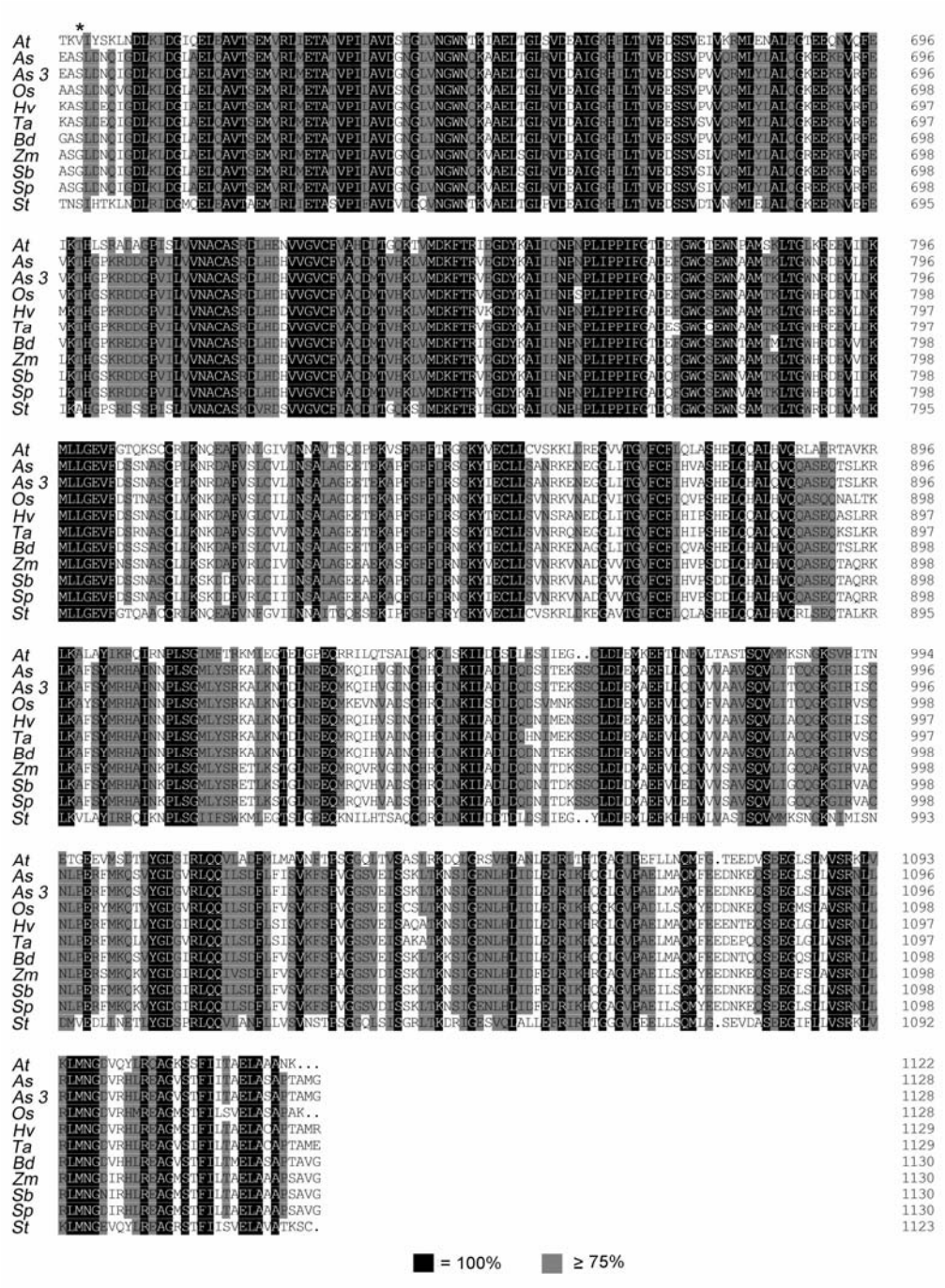

图1 单子叶和双子叶植物中phyA蛋白的多序列对比

At: 拟南芥phyA; *As*: 燕麦phyA; *As3*: 燕麦phyA 3型; *Os*: 水稻phyA; *Hv*: 大麦亚种phyA; *Ta*: 小麦phyA 1型; *Bd*: 二穗短柄草phyA 3型; *Zm*: 玉米phyA; *Sb*: 高粱phyA; *Sp*: 拟高粱phyA; *St*: 马铃薯phyA。*表示燕麦phyA中已鉴定的磷酸化位点Ser8、Ser18和Ser598

Figure 1 Multiple sequence alignments of monocot and dicot phyA proteins

At: *Arabidopsis thaliana* phyA; *As*: *Avena sativa* phyA; *As*: *Avena sativa* phyA type 3; *Os*: *Oryza sativa* phyA; *Hv*: *Hordeum vulgare* subsp. *vulgare* phyA; *Ta*: *Triticum aestivum* phyA type 1; *Bd*: *Brachypodium distachyon* phyA type 3-like; *Zm*: *Zea mays* phyA; *Sb*: *Sorghum bicolor* phyA; *Sp*: *Sorghum propinquum* phyA; *St*: *Solanum tuberosum* phyA. * indicates phosphorylation sites at Ser8, Ser18 and Ser598 identified in oat phyA

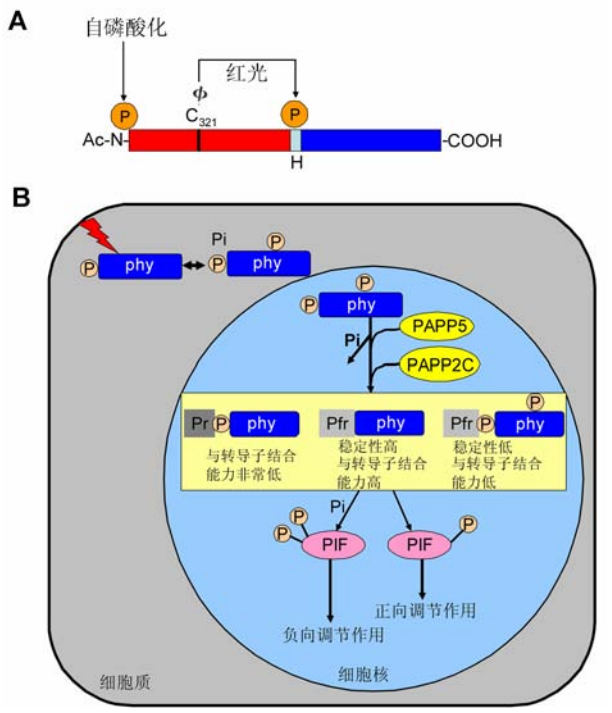

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图2 光敏色素信号通路的磷酸化修饰模式图(Ryu et al., 2005; Galvão et al., 2012)

(A) 燕麦phyA结构域和磷酸化位点。N端结构域(N)和C端结构域(COOH)用矩形表示，Φ表示植物后胆色素，它与氨基酸Cys321 (C321)共价连接。中间的小矩形表示铰链区(H)，中间箭头指示的位置为铰链区域一个磷酸化位点Ser598。目前已经报道燕麦phyA有3种翻译后修饰: N-乙酰化(Ac-N)，植物后胆色素连接到phyA 321位的半胱氨酸上以及磷酸化N端区域的第7位丝氨酸。光敏色素的自磷酸化位点在N端区域(Lapko et al., 1999); **(B)** 光促使无活性的光敏色素转变为有活性的光敏色素，从而开启了光敏色素调节的光信号，有活性的光敏色素可以自磷酸化以及被一些光敏色素相关的激酶磷酸化。磷酸化的光敏色素会被一些磷酸酶(PAPP5和PAPP2C)去磷酸化。有活性的光敏色素在N端和铰链区被去磷酸化从而缓解了磷酸化介导的去稳定作用，进而与下游信号分子结合能力增强，而没有磷酸化的有活性的光敏色素反之。此外，磷酸化的没有活性的光敏色素与下游信号分子结合能力非常低。这些机制导致下游PIF存在2种状态，一种是PIF被光敏色素磷酸化从而负向调控光信号; 另一种是光敏色素由于被去磷酸化所以不能磷酸化PIF，最终导致正向调节光信号。

Figure 2 A proposed model of phosphorylation modification in the phytochrome signaling pathway (Ryu et al., 2005; Galvão et al., 2012)

(A) Domain structure and phosphorylation sites in oat phyA. The N-terminal (N) and C-terminal domains (COOH) are shown by rectangles. Φ indicates phytochromobilin, covalently attached to Cys321 (C321). The middle of the small rectangle represents “hinge region (H)”, the phosphorylation site at Ser598 is shown by arrow in the middle. Three types of posttranslational modification of oat phyA have been reported previously: N-acetylation (ac-N), phytochromobilin ligation to Cys321 and phosphorylation in the N-terminal region at Ser7. Phosphorylation at the N-terminus was suggested to be the site of phytochrome “autophosphorylation” (Lapko et al., 1999); **(B)** Light triggers photoconversion of the Pr-phytochromes to the Pfr-phytochromes, which initiates the phytochrome-mediated photosignaling. Pfr-phytochromes are phosphorylated by their intrinsic kinase activity as well as by phytochromes-associated kinase(s) and are reversibly dephosphorylated by phosphatase, such as, PAPP5, PAP-P2C. The Pfr-phytochromes dephosphorylates in the N-terminal extension and the hinge region is relieved from phosphorylation mediated destabilization, exhibiting a high affinity to signal transducers. However, the unphosphorylated Pfr-phytochromes were not. Furthermore, the phosphorylated Pr-phytochromes possess a very lower affinity toward signal transducers. These mechanisms lead to the existence of two state of PIF. One is PIF was phosphorylated by phytochromes results in the negative regulation of light signaling. Another is phytochromes were dephosphorylated and could not phosphorylate PIF, which results in the positive regulation of light signaling.

时光敏色素的磷酸化还影响其与PIF3的相互作用(Kim et al., 2004)。phyA铰链区位点处于去磷酸化时与下游转导子的结合比较强，当该位点被磷酸化后，这种结合能力显著降低(图2)，说明光敏色素的铰链区作为一个磷酸化信号调节位点调控phyA与信号分子之间的相互作用(Kim et al., 2004)。

在拟南芥中已发现phyA可以被磷酸化并且phyA的磷酸化可以调节其与COP1/SPA1和FHY1/FHY3的聚集(Saijo et al., 2008)，但是这些功能的重要性还不是很清楚。与燕麦和水稻phyA类似，拟南芥phyA N端区域对于信号的传递也非常重要(Cherry et al., 1992; Jordan et al., 1997)，但是分析拟南芥phyA N端突变体并没有发现磷酸化可以调节燕麦phyA信号。在拟南芥中，将野生型燕麦*PHYA*转入*phyA-201*突变体中，发现它可以恢复*phyA*的缺陷表型，将突变的燕麦*PHYAser598Ala*转入*phyA-201*突变体，发现转基因植株表现出对光非常敏感，说明光敏色素在铰链区Ser598位点发生磷酸化存在一种抑制机制(Kim et al., 2004)。但是目前还没有证据证实拟南芥中Ser598位点的具体功能，所以还需要深入研究拟南芥phyA磷酸化对其信号通路的影响。

目前，对于植物phyA是否磷酸化其它蛋白还有相当多的争议。体外证据表明，phyA可以磷酸化

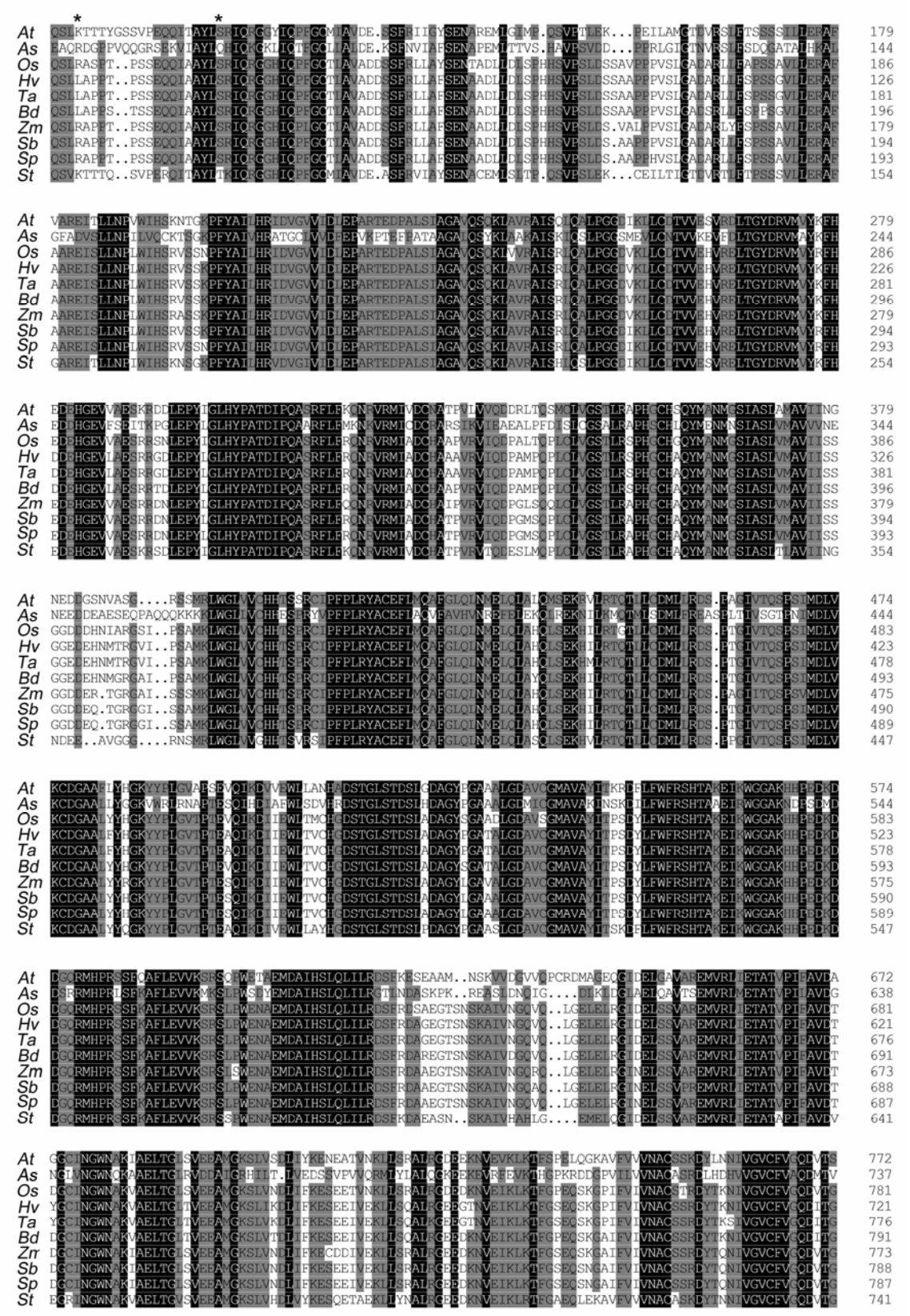

图3

Figure 3

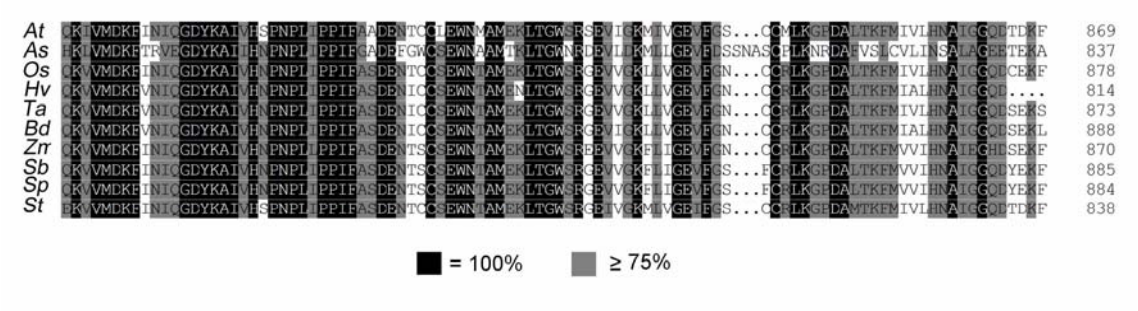

图3 单子叶和双子叶植物中phyB蛋白的多序列对比

At: 拟南芥phyB; *As*: 燕麦phyB; *Os*: 水稻phyB; *Hv*: 大麦亚种phyB; *Ta*: 小麦phyB; *Bd*: 二穗短柄草类phyB; *Zm*: 玉米phyB 2; *Sb*: 高粱phyB; *Sp*: 拟高粱phyB; *St*: 马铃薯phyB。*表示拟南芥phyB中已鉴定的磷酸化位点Ser86和Tyr104

Figure 3 Multiple sequence alignments of monocot and dicot phyB proteins

At: *Arabidopsis thaliana* phyB; *As*: *Avena sativa* phyB; *Os*: *Oryza sativa* phyB; *Hv*: *Hordeum vulgare* subsp. *vulgare* phyB; *Ta*: *Triticum aestivum* putative phyB; *Bd*: *Brachypodium distachyon* phyB-like; *Zm*: *Zea mays* phyB 2; *Sb*: *Sorghum bicolor* phyB; *Sp*: *Sorghum propinquum* phyB; *St*: *Solanum tuberosum* phyB. * indicates phosphorylation sites at Ser86 and Tyr104 identified in Arabidopsis phyB

cryptochrome1 (Cry1)、phytochrome kinase substrate 1 (PKS1)和auxin/indole-3-acetic acid (Aux/IAA), 但是还没有在植物中证明这些结论(Ahmad et al., 1998; Fankhauser et al., 1999; Colón-Carmona et al., 2000)。对于拟南芥phyA的磷酸化作用, 最近研究表明, 一个光诱导phyA入核的关键调节子FHY1, 在R/FR光转换过程中被磷酸化, 在红光下磷酸化FHY1需要phyA参与(Shen et al., 2009; Chen et al., 2012), 同时磷酸化FHY1降低了有活性形式的phyA入核速率及phyA与HY5和PIF3的相互作用, 结果导致phyA在远红光下转录复合体的组装失活(Yang et al., 2009; Chen et al., 2012)。这些结果说明, phyA磷酸化其它蛋白对其信号传递非常重要。

目前, 有研究表明, 一些蛋白可以与phyA相互作用并去磷酸化phyA, 这些蛋白包括flower-specific phytochrome-associated protein phosphatase (FyPP) (Kim et al., 2002)、phytochrome-associated protein phosphatase type 2C (PAPP2C) (Phee et al., 2008)和phytochrome-associated protein phosphatase 5 (PAPP5) (Ryu et al., 2005)。转基因植株超表达*FyPP*后在开花和下胚轴伸长方面增强了光敏色素活性, 抑制*FyPP*表达则导致转基因植株中光敏色素活性降低(Kim et al., 2002)。phy与PIF3在细胞核内相互作用并将PIF3磷酸化从而负向调节光信号, 而PAPP2C可以与phyA相互作用并将phyA去磷酸化, 这个互作优先于phyA与PIF3的结合, 从而导致PIF3不能被磷酸化, 最终对光信号通路起正调节作用(Phee et al., 2008)。PAPP5是一类5型磷酸酶, 其与燕麦phyA结合形成一类特殊的构象, 可以对phyA的3个已知磷酸化位点进行去磷酸化, 从而调节幼苗的去黄化, 影响phyA蛋白的稳定性, 并且去磷酸化的燕麦phyA增加了其与NDPK2和PIF3的结合能力。在拟南芥中超表达磷酸酶PAPP5可导致其对远红光敏感, *papp-5*突变体则表现出对远红光敏感性降低(Ryu et al., 2005)。这些发现证明, phyA的去磷酸化在光敏色素调节的光信号通路中起重要作用。

2.2 phyB的磷酸化与去磷酸化作用

相比phyA, 目前关于phyB磷酸化的研究较少。研究发现, *PAPP5*无义突变体*papp-5*对红光敏感, PAPP5磷酸酶在体外pull-down实验中可以与phyB相互作用, 并且在转基因植株中phyB与PAPP5共定位在光体中。进一步实验证明, phyB作为PAPP5的底物, PAPP5在体外可以将phyB去磷酸化(Ryu et al., 2005)。随后有许多报道表明, phyB与另一类磷酸酶PAPP2C在体外也可以相互作用, 并且这个磷酸酶优先与有活性的phyB相互作用, 在红光照射下PAPP2C与phyB共定位于细胞核内, 在黑暗条件下则无此现象。自磷酸化的phyB可以被PAPP2C去磷酸化, 并且缺失突变体*papp2c*对红光响应降低(Phee et al.,

2008)。这些结果说明，可逆磷酸化在phyB调节的信号通路和光形态建成中起重要作用。

最近，Ferenc Nagy实验室通过质谱实验发现了拟南芥phyB的磷酸化位点，证明了phyB在植物体内能被磷酸化。无论在黑暗或红光条件下，phyB N端的Ser86位点都可被磷酸化。通过比对单子叶和双子叶植物中phyB的Ser86位点，发现该位点在不同物种中是高度保守的(图3)。通过研究模拟磷酸化植株*phyB*^{Ser86Asp}*-YFP*，发现其对红光比较敏感，但是这种敏感度的差异，仅在低强度的红光下比较明显。在phyB中Ser86替换为Ala或Asp并没有改变这些融合蛋白的稳定性。研究还发现在体外非饱和光照条件下，模拟磷酸化的phyB与PIF3的结合能力降低(图2)。同时，*phyB*^{Ser86Asp}*-YFP*中光诱导光体在细胞核内的积累和积聚减少了(Medzihradszky et al., 2013)。目前，还不清楚是由于phyB的自磷酸化还是由于未知激酶的作用使phyB磷酸化，从而显著改变phyB的亚核分布。上述结果表明，磷酸化光受体可能会调节光体的形成(Medzihradszky et al., 2013)。

最近的研究还发现，在phyB的N端存在一个含有23个氨基酸残基的区域，叫做PCSM (phosphorylation cluster of signaling modulation)区域，当植物受到光照后，PCSM区域中许多氨基酸被磷酸化，这些位点中除了丝氨酸和苏氨酸外，还包括一个酪氨酸位点(Tyr104) (Nito et al., 2013)，该位点在不同物种中都完全保守(图3)。研究发现在拟南芥中模拟磷酸化的phyB^{Tyr104Glu}不能恢复*phyB*的相关表型，并且该突变蛋白没有phyB活性。phyB^{Tyr104Glu}不能与信号分子PIF3相互结合，也不能形成稳定的光体。在植株中稳定表达不能磷酸化的phyB^{Tyr104Phe}，会使植株表现出对光超敏感。在光环境的急剧变化过程中，由于植物适应环境对其存活非常关键，所以酪氨酸磷酸化phyB作为在光信号机制中新发现的一种翻译后修饰，对研究光敏色素信号通路有非常重要的意义(Nito et al., 2013)。

3 PIFs的磷酸化修饰

3.1 PIFs转录因子的功能

PIFs是一类bHLH转录因子，负向调节光形态建成(Leivar and Quail, 2011)。1998年，Peter Quail实验室发现了PIFs家族成员PIF3，这为全面开展PIFs在光形态建成中的作用研究奠定了很重要的基础(Ni et al., 1998)。目前，鉴定到的PIFs包括PIF3、PIF1/PIL5、PIF4、PIF5/PIL6和PIF7，它们可以与光调节基因的G-box区域结合，作为转录激活子或转录抑制子起作用(Huq and Quail, 2002; Huq et al., 2004; De Lucas et al., 2008; Leivar et al., 2008b, 2009; Moon et al., 2008; Hornitschek et al., 2009)。PIFs在光敏色素介导的光响应中起着不同的作用。在去黄化过程中，PIF1、PIF3、PIF4、PIF5和PIF7抑制植物的下胚轴伸长(Huq and Quail, 2002; Fujimori et al., 2004; Huq et al., 2004; Khanna et al., 2004; Oh et al., 2004; Al-Sady et al., 2008; Lorrain et al., 2009)。在叶绿体发育过程中，PIF1、PIF3和PIF5抑制叶绿体的发育，下调编码叶绿素关键合成酶基因的表达(Huq et al., 2004; Moon et al., 2008; Leivar et al., 2009; Shin et al., 2009; Stephenson et al., 2009)。另外，PIF1还下调类胡萝卜素的表达(Toledo-Ortiz et al., 2010)。*pifQ*突变体在黑暗条件下表现出去黄化的表型(Hu et al., 2009; Leivar et al., 2009)。这些结果说明，PIFs家族在黑暗条件下可以促进暗形态建成，抑制光形态建成，且在功能上存在冗余(Leivar et al., 2008b; Shin et al., 2009)。

3.2 PIF蛋白磷酸化修饰的功能

PIF蛋白的N端区域包含光敏色素的作用区域，C端包含bHLH DNA结合区域和二聚化区域。N端的保守区域，称为激活phyB结合区域(the active phytochrome B-binding region, APB)，该区域对于与phyB的特异性结合是必需的(Khanna et al., 2004)。PIF1和PIF3包含一个独立的区域称为激活phyA结合区域(the active phytochrome A-binding region, APA)，该区域对与phyA的结合是必需的(Al-Sady et al., 2006; Shen et al., 2008)。PIF1和PIF3能与光敏化的phyA和phyB结合(Al-Sady et al., 2006; Shen et al., 2008)，而PIF4、PIF5、PIF6和PIF7只能与phyB结合(Ni et al., 1998; Huq et al., 2004; Khanna et al., 2004; Leivar et al., 2008a)。光激活phys后，phys积聚在细胞核内与PIFs蛋白相互作用，使得PIFs被磷酸化并通过泛素蛋白酶体途径迅速降解(Al-Sady et al., 2006)。光诱导PIFs磷酸化和降解需要与有活性的

phys直接相互作用(Al-Sady et al., 2006; Shen et al., 2008)。PIF3与光敏化的phys共定位形成光体也需要与有活性的phys直接相互作用(Bauer et al., 2004; Al-Sady et al., 2006; Chen and Chory, 2011)。突变的PIF3蛋白不能与phyA和phyB结合，所以检测不到phys诱导的PIF3磷酸化以及降解(Al-Sady et al., 2006)。光诱导PIF3磷酸化可以通过检测PIF3迁移率的变化来证明(Al-Sady et al., 2006)。这些结果表明，光敏化的phys是介导PIF3磷酸化并最终通过泛素化途径降解的主要因子。目前，已经证明PIF1、PIF4和PIF5在光诱导降解之前都会被磷酸化修饰(Shen et al., 2007; Lorrain et al., 2008; Shen et al., 2008)。因此，光诱导的PIFs蛋白磷酸化是信号从光敏化的phys传递到转录因子PIFs最主要的生物学机制。然而，目前还不清楚PIFs的磷酸化是哪个激酶在起作用。最近报道证明，PIF1可以被CK2 (casein kinase II)以及可能没有发现的激酶磷酸化(Bu et al., 2011a)。PIF4可以与BZR1 (brassinazole-resistant 1)相互作用，它们在响应BR、黑暗以及热激时相互独立地调控细胞伸长，并且可以共同调控一些光合激素响应的基因，进而证明BZR1-PIF4相互作用，从而控制了一个核心的转录网络，使得植株生长受到甾醇和环境因子的共同调控(Oh et al., 2012)。但是对于PIF4通过什么机理与BR信号共同调控下胚轴伸长尚不清楚。有报道显示BR信号通路中BIN2 (brassinosteroid-insensitive 2)可以将PIF4磷酸化，这就找到了PIF4的一个激酶。研究发现，转基因植株PIF41A中，PIF4与BIN2结合区域中的关键位点突变，结果导致PIF4不能被BIN2磷酸化，最终使得植株中PIF4的磷酸化和降解都明显受到抑制。BR通过抑制BIN2活性拮抗地参与光信号，从而调节PIF4的去稳定性。同时，PIF4的磷酸化对于其调节周期性的下胚轴生长也非常重要(Bernardo-Garcia et al., 2014)。但截至目前，已知的可以磷酸化PIFs的激酶还很少，所以尚需要进一步研究。

为了更好地分析信号分子从光敏化的phy到PIF转录因子转移过程的分子本质，2013年，Quail实验室找到了PIF3的磷酸化位点，并将该位点定点突变，结果发现当13个磷酸化位点全部突变为Ala时，突变的PIF3 (PIF3-A13)表现出明显光诱导的迁移率降低以及光诱导的降解变慢(Ni et al., 2013)。将其余磷酸化位点全部突变后，PIF3迁移率降低且降解抑制作用更明显。同时检测这些转基因植株的泛素化水平，结果表明泛素化修饰蛋白的积累需要光诱导的PIF3磷酸化。PIF3磷酸化位点的突变不能影响其与phyB的结合，也不影响其与特异的DNA序列结合。经过红光诱导后，PIF3与phyB在亚核结构中共定位形成核斑点，检测PIF3磷酸化位点突变转基因植株*PIF3-A14*、*PIF3-A20*和*PIF3-A26*，发现明显存在光体定位，但*PIF3-A14*和*PIF3-A20*中PIF3形成的光体消失的速率变慢，表明光诱导的磷酸化对于PIF3光体定位不是必需的。检测模拟磷酸化植株*PIF3-D6*和*PIF3-D19*的磷酸化，发现这些植株在黑暗条件下已经发生磷酸化，并且部分蛋白发生降解，进一步说明PIF3磷酸化对于其降解是必要的(Ni et al., 2013)。

单基因突变体*pif*以及*PIF*超表达突变体幼苗均表现出复杂的形态模式(Leivar and Quail, 2011)。*pif*突变体对持续红光照射非常敏感(Huq and Quail, 2002; Kim et al., 2003; Fujimori et al., 2004; Monte et al., 2004; Khanna et al., 2007; Nozue et al., 2007; de Lucas et al., 2008; Leivar et al., 2008a; Lorrain et al., 2008, 2009; Shin et al., 2009)。这些突变体对红光的响应，在某种程度上是由于phyB的大量积累，从而导致了它们对光的超敏感性。phyB大量积累主要是因为PIF蛋白降解反馈调节光敏化的phyB降解，而在突变体中这种反馈作用降低，使phyB的降解减少，最终导致phyB的积累(Khanna et al., 2007; Al-Sady et al., 2008; Leivar et al., 2008a, 2012)。通过检测转基因植株*PIF3-WT*、*PIF3-A14*、*PIF3-A20*和*PIF3-A26*体内的phyB含量，发现PIFs反馈调节phyB不仅与phyB相互作用的PIFs含量有关，而且与体内光诱导PIFs降解的速率有关(Ni et al., 2013)。目前，光诱导PIF3磷酸化并最终影响其降解的机制尚不清楚(Bu et al., 2011a, 2011b)。最新研究表明，PIF的磷酸化可以招募一种光响应的LRB (light-response bric-a-brack/tramtrack/broad (BTB)) E3泛素连接酶结合到PIF3和phyB的复合物上。招募的LRBs可以促使PIF3和phyB泛素化以及降解。这些结果揭示了光受体及其所调节的信号分子之间存在一种相互保护且相互抑制的信号传递和信号终止机制，该机制与以往的报道不同。然而，尽管在LRB的突变体*lrb123*中光诱导的phyB泛素化以及降解下游PIF基本消除，但是PIF3的泛素化

和降解只是变慢并没有完全消失，说明可能还有一些未知E3连接酶在调控PIF3的降解过程中与LRBs存在功能冗余(Ni et al., 2014)。

4 展望

过去十多年中，光敏色素信号机制的研究已经取得了巨大的进展。然而，对于光敏色素调控信号网络的本质和范围所知甚少。在光敏色素信号通路中，磷酸化修饰作为一种重要的机制还未被完全解析清楚。光敏色素除了自磷酸化外，是否还有激酶参与其去磷酸化；光敏色素的去磷酸化如何影响其信号传递和功能等；诸如此类的问题还有待解决。同时，光敏色素需通过磷酸化PIF蛋白传递信号，PIF自身磷酸化对于其降解至关重要，但具体分子本质目前还不是很清楚。此外，PIF蛋白的去磷酸化到现在还一无所知。未来的挑战将是寻找调控光敏色素以及PIF蛋白磷酸化水平的激酶和磷酸酶。

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Research Progress in Phosphorylation Modification of Phytochrome Signaling

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Abstract Light is the unique source of energy for plants. Plants have evolved a variety of photoreceptors to sense light information. The elements in the light signaling pathway are mainly regulated by several post-translational modifications such as phosphorylation and dephosphorylation. Photochromes, the known auto-phosphorylating serine/threonine kinases, can be dephosphorylated by a few protein phosphatases. Investigation of the autophosphorylation sites in phytochrome A (phyA) and phytochrome B (phyB) has revealed that the autophosphorylation of phy is essential for their function and plays a significant role in regulating phytochrome-mediated signaling. The phosphorylation of phytochrome-interacting factor (PIF) induced by the light-activated photoreceptor is critical for PIF degradation and photomorphogenesis initiation. This review focuses on the recent progress in understanding phosphorylation modification in phytochrome signaling, providing valuable information for further research in this field.

Key words phytochrome, PIF, phosphorylation modification

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