拟南芥斑叶突变体及其斑叶形成机制研究进展

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摘要: 拟南芥斑叶突变体是研究叶绿体发育机理的理想材料, 因斑叶严重程度受到遗传与环境因子以及它们之间互作的影响。在同一遗传背景下, 为什么叶片会分化出绿色和白色或者黄色的斑块一直是人们关心的重要科学问题。研究表明大部分通过遗传筛选获得的斑叶抑制子直接参与叶绿体基因表达的各个生化过程, 对斑叶及其抑制子的研究加深了我们对叶绿体发育的分子机制的认识。

关键词: 斑叶; 叶绿体发育; 抑制子; 叶绿体基因表达; 拟南芥

斑叶是指同一叶片上绿色和黄/白色区域并存的表型, 叶绿体在绿色区域(green sector)发育正常, 而在黄/白色区域(yellow/white sector)发育异常(图1)。黄/白色区域的大小通常与光强和温度等环境因子有关(Liu等2010b; Yu等2007; Aluru等2006)。斑叶在单子叶植物中往往表现为绿与白/黄条状镶嵌。斑叶现象广泛存在于高等植物中, 因遮荫、病菌侵染、营养缺陷等引起的斑叶是不能遗传的, 而由核基因、线粒体基因或叶绿体基因突变所引起的则可稳定遗传(Rodermel 2002)。自然界中, 斑叶产生的生物学意义及其进化上的价值并不清楚。在二十世纪初斑叶表型被用作遗传标记, 在非孟德尔遗传规律的发现过程中起了重要作用。目前, 斑叶性状广泛应用于花卉育种、园林绿化, 拓展了品种多样性; 在基础理论研究中, 斑叶植物是研究叶绿体发育机制的理想材料(Yu等2007)。本文着重介绍模式植物拟南芥中的斑叶突变体及其形成机制。

图1 斑叶突变体表型

Fig.1 Leaf variegation phenotype

1 斑叶突变体的特征

现有的研究显示斑叶上的黄/白区域是由叶绿体发育受阻引起的, 但细胞是活的(Rodermel 2002)。在很多情况下, 斑叶是由单个隐性基因突变引起的。在斑叶的黄/白交接区, 能观测到一些细胞中含有几个正常叶绿体。这与早期报道的很少见到两种质体同时存在于一个细胞的结果有所不同(Wu等2011; Wetzel等1994)。因此, 很有必要从细胞示踪的角度研究白色或黄色区的细胞是否来自于一个细胞。

2 拟南芥斑叶突变体

迄今为止, 拟南芥中已报道的由核基因突变引起的斑叶突变体包括*im* (*immutans*)、*var1* (*variegated 1*)、*var2* (*variegated 2*)、*var3* (*variegated 3*)、*thf1* (*thylakoid formation1*)、*chm/msh1* (*chloroplast mutater/muts homolog 1*)、*why* (*whirly*) *1 why3*双突变、*msl* (*mechanosensory-like proteins*) *2 msl3*双突变、*atd2*、*cla1*等。这些基因克隆及其功能研究有助于我们对斑叶形成机理的深入了解。

2.1 *im*突变体

*im*突变体的报道已有40多年了(Rédei 1973)。*IM*编码一个定位于叶绿体的末端氧化酶(plastid terminal oxidase, PTOX), 其生化功能类似于线粒体内膜上的交替氧化酶(alternative oxidase, AOX) (Carol等1999; Wu等1999), 负责将电子从泛醌传递到氧分子而生成水(Vanlerberghe和McIntosh 1997), 起着平衡氧化还原的作用(Giraud等2008; McDonald 2008)。体外实验证明重组IM蛋白确实具有质体醌氧化酶活性, 这种活性能被AOX的抑制剂没食子酸丙酯(*n*-propylgallate)所抑制(Josse等2000), 且质体醌是IM特异性的底物(Josse等2003)。

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PTOX的起源被认为是发生在内共生事件之后(McDonald等2011)。PTOX结合于类囊体膜基质片层, 其活性位点位于类囊体膜基质侧(Lennon等2003; Joët等2002; Berthold等2000)。关于PTOX的生理功能, 早期的报道认为PTOX参与调控胡萝卜素的合成, 因为在*im*突变体的白色区域积累胡萝卜素的前体产物八氢番茄红素(Rochaix 2011), 推测PTOX是八氢番茄红素脱氢酶(PDS)的耦联因子(McDonald等2011; Aluru等2006; Sakamoto 2003; Carol等1999; Wu等1999)。除了参与胡萝卜素合成途径, IM作为氧化质体醌的末端氧化酶, 参与叶绿体发育与回绕PSI的循环电子传递等过程中(Foudree等2012; Yu等2007; Aluru等2006; Joët等2002; Cournac等2000)。在水稻中PTOX突变导致分蘖数增加、植株矮化、叶片产生花斑等表型(Tamiru等2014)。

为什么*im*突变体依然还能产生正常的叶绿体呢？一种可能是来自电子传递链中质体醌库下游的还原型物质或其它质体氧化酶可以补偿IM的功能, 降低质体的光氧化风险。质体能够忍受一定程度的氧化胁迫(阈值), 而氧化胁迫高于某个阈值时叶绿体发育就会受阻。每个质体的阈值因其补偿活性物质的多少或光保护能力的强弱而不同, 从而产生所谓的质体异质性, 最终形成宏观上的斑叶表型(Foudree等2012; Rosso等2009; Yu等2007; Aluru等2006)。另一种解释是在叶绿体早期发育时期, 激发压力(excitation pressure)的增加导致斑叶的产生。激发压力是指光系统II电子受体QA还原状态的一个相对值, 当激发压力高于一定阈值时会导致质体光氧化, 而低于阈值时可以发育成正常的叶绿体(Foudree等2012; Rosso等2009)。

2.2 *var1*和*var2*突变体

*var1*和*var2*的子叶与野生型一样, 但它们的真叶呈现斑叶表型, 其严重程度受到光强等环境因子的影响(Sakamoto等2002; Chen等2000)。*VAR1*和*VAR2*分别编码定位于叶绿体的、属于AAA$^+$ (ATPase associated with diverse cellular activities)家族的FtsH金属蛋白酶FtsH5和FtsH2。FtsH蛋白包含N端跨膜结构域、保守的ATP水解酶(Walker A和Walker B)和蛋白水解酶结构域。研究表明与叶绿体发育相关的FtsH复合体是由FtsH1和FtsH5 (A型)与FtsH2和FtsH8 (B型)组成的异源六聚体, 同时缺失A型或者B型中两个成员则导致植物白化致死(Putarjunan等2013; Liu等2010b; Zaltsman等2005a; Sakamoto等2003), 而同组内两个成员的功能是冗余的(Putarjunan等2013; Liu等2010b; Zaltsman等2005a; Yu等2004, 2005), 说明FtsH复合体是叶绿体发育所必需的。FtsH复合体定位于类囊体膜上, 通过降解遭受光破坏的D1蛋白的23 kDa片段参与PSII的修复过程(Kato等2009, 2012; Lindahl等2000)。

虽然FtsH调控叶绿体发育的机理迄今还不清楚, 但是研究结果显示*var2*白色区域的质体中活性氧(reactive oxygen species, ROS)积累(Kato等2009, 2012), 分化停滞在类囊体发育的早期, 光合作用相关的基因表达下调(Sakamoto等2009; Kato等2007)。有意思的是蛋白水解酶活性位点突变的FtsH2^{H488L} (即水解酶结构域的488位活性位点突变导致水解活性丧失), 可以恢复*var2*的斑叶表型和*var2 ftsh8*双突变的致死表型, 说明B型FtsH成员的蛋白水解酶活性对FtsH的功能不是必需的; 但FtsH2^{H488L}不能完全恢复*var1 var2*双突变体的斑叶表型, 说明FtsH2的整体表达水平对叶绿体发育的重要性(Zhang等2010)。这些结果辩证地解释了FtsH亚基的活性与表达量对整个复合体功能的重要性。

2.3 *thf1*突变体

高等植物中的THF1是一个蓝藻Psb29的同源蛋白, 定位于叶绿体, 由单拷贝基因编码, 在光合放氧生物中高度保守。除了真叶外, *thf1*突变体的子叶也有花斑, 而*im*、*var1*和*var2*突变体的子叶则呈现绿色。在*thf1*黄/白色区域中, 质体缺乏完整的类囊体膜结构, 并且有很多膜泡的积累, 推测THF1可能在类囊体膜形成过程中起着囊泡运输和组装的作用(Wang等2004)。虽然绿色区域的叶绿体发育与野生型之间没有明显的差异, 但*THF1*突变导致强光下生长减慢、PSII活性降低, 这与蓝藻*psb29*突变体的表型是一致的(Keren等2005)。因此, THF1/Psb29的基本功能可能是一样的。

THF1在叶绿体被膜、基质、类囊体膜和质体管(stromules)中均有分布, 暗示其参与叶绿体中的多种生物学过程。Huang等(2006)发现THF1能与质膜异三元G蛋白的alpha亚基(AtGPA1)相互作

用, 参与糖信号转导途径。小麦中THF1同源蛋白ToxABP1与小麦黄斑叶枯病菌(*Pyrenophoratritici-repentis*)产生的类蛋白毒素PtrToxA相互作用(Manning等2007), 而番茄中*THF1*同源基因*SlALC1*突变对丁香假单胞菌(*Pseudomonas syringae*) DC3000侵染或冠菌素(coronatine, COR)处理超敏感, 细胞死亡加速, 这个结果在拟南芥*thf1*突变体中也得到了验证。因此, THF1在冠菌素信号途径和疾病反应过程中也起作用(Wangdi等2010)。这与*thf1*突变体中花青素含量积累是由于JA含量增加引起的结果一致(Gan等2014)。最近的研究发现THF1突变影响叶片衰老等过程中的叶绿素降解过程(Huang等2013), 遗传与生化等数据显示THF1直接与LHCII互作, 且其互作受pH的调控, 参与PSII-LHCII复合体的动态调控(Huang等2013)。但是, THF1的生化功能及其调控众多生理过程的分子机理有待阐明。

2.4 *chm*或者*msh1*以及*why1 why3*突变体

*CHM*编码一个与大肠杆菌错配修复及DNA重组相关的MutS类同源蛋白(Abdelnoor等2003)。酵母中MutS的同源蛋白MSH1参与线粒体基因组的错配修复, 酵母*msh1*突变体线粒体基因组发生重排, 功能异常, 呼吸作用受损(Abdelnoor等2003; Sakamoto 2003)。拟南芥*chm*突变体由Rédei (1973)通过筛选EMS突变体库获得, 表现为隐性基因控制的母性遗传(Rodermel 2002)。上世纪90年代, 研究发现*chm*突变体中线粒体基因组发生了重排(Martínez-Zapater等1992), 存在一种特殊构象的线粒体DNA (mtDNA)。进一步分析表明, 野生型特异性的和*chm*特异性的mtDNA构象在野生型和*chm*植株中均存在, 只是比例不同。因此, CHM对于维持正常构象的mtDNA和/或清除异常构象的mtDNA是非常重要的(Sakamoto等1996, 2003)。还发现突变体中大量积累的异常线粒体构象导致部分线粒体基因(如*rps3*、*rpl16*)不能正常表达, 从而导致线粒体功能的异常(Sakamoto等1996)。后来的研究发现CHM/MSH1 (MutS HOMOLOG1)不仅定位于线粒体也定位于叶绿体, 而且证明了斑叶表型是由定位于叶绿体的MSH1决定的, 而与线粒体定位的MSH1无关(Xu等2011)。

在植物中, 还发现了另外一类蛋白质Whirly (Why)参与线粒体和质体基因组高效复制、重组和修复途径, 即DNA replication, recombination, and repair (DNA-RRR)。Why是植物中广泛存在的、与单链DNA结合蛋白, 其成员参与抗病、端粒长度平衡等很多生物学过程(Yoo等2007; Desveaux等2005)。Why1和Why3靶向叶绿体(Krause等2005), 双突变体*why1 why3*产生母性遗传的斑叶表型。在黄/白色区域中, 质体因DNA发生非特异重组而产生环形或首尾相连的连接子的频率大幅增加, 这些连接子不能进行正常的转录和翻译, 从而影响了叶绿体的功能(Maréchal等2009)。

以上结果说明叶绿体基因组的稳定和质体发育密切相关。植物中可能还存在其他蛋白调控细胞器基因组的稳定、复制和修复等过程, 这些基因的突变也将导致叶绿体发育异常, 产生斑叶表型。

2.5 *msl2 msl3*双突变体

MSL是类似于细菌MscS (mechanosensitive channel of small conductance)的压力感受离子通道, 在拟南芥中有10个成员, 其中MSL2和MSL3的同源性很高。结构解析结果显示大肠杆菌MscS复合体是一个七聚体, 每个亚基N端的3个跨膜结构域组成离子通道, C端结构域互作形成一个预滤器。互补实验证明拟南芥的MSL3能够恢复大肠杆菌渗透胁迫敏感突变株MJF465 (MS离子通道活性缺失)的表型(Levina等1999)。*msl2*与*msl3*的单突变体没有明显的表型缺陷, 但双突变体从第三或第四片真叶开始出现淡绿色区域(斑叶), 斑叶在茎身叶上特别明显。叶片结构观察发现*msl2 msl3*双突变叶肉细胞变大、细胞排列松散、细胞形状也异常。叶绿体超微结构分析表明*msl2 msl3*双突变中的叶绿体明显大于野生型, 但其类囊体膜结构正常。因此, 推测MSL2和MSL3是维持叶绿体大小和形状所必需的。研究还发现, MSL2和MSL3与控制叶绿体分裂的AtMinE共定位, 但不直接影响AtMinE的功能, 提示MSL2和MSL3在叶绿体分裂过程中起作用(Haswell和Meyerowitz 2006)。

MSL2和MSL3被认为可能通过感受叶绿体被膜压力、释放质体的离子从而维持质体的大小和形状, 同时可能参与质体分裂过程中压力的释放, 从而维持质体正常的分裂(Haswell和Meyerowitz 2006)。关于MSL2和MSL3调控叶绿体发育的分子机理还需要进一步研究。

2.6 其它斑叶突变体

叶绿体也是众多化合物的代谢中心。除了光合作用外, 氨基酸、核酸、萜类化合物以及植物激素等重要物质的合成均在叶绿体中进行。研究表明这些代谢途径的缺陷与叶绿体发育密切相关。完全抑制这些代谢途径往往导致植株白化的表型, 而部分抑制会产生斑叶的表型。例如嘌呤合成途径中的第1个酶——谷氨酰氨磷酸核糖焦磷酸转酰氨酶2 (Atase2/CIA1/DOV1)缺失导致真叶呈现叶脉绿色而叶肉黄色或淡绿的网状斑叶以及细胞分裂素含量与细胞数显著减少等表型(Yang等2015; Rosar等2012; Hung等2004); 萜类化合物合成主干途径(MEP)上DXP合成酶(1-deoxy-D-xylulose 5-phosphate synthase)活性降低的突变体(*cla1*/*lvr111*/*chs5*)中, 类异戊二烯的含量与叶绿体发育缺陷的严重程度直接关联(Yu等2007; Crowell等2003; Araki等2000; Mandel等1996)。

VAR3是一个位于叶绿体基质的含锌指结构的蛋白质, 其突变导致真叶呈现花斑表型, 黄色区域的栅栏细胞大量减少, 叶绿体发育延迟(Næsted等2004)。早期的研究认为*var3*突变体的表型与叶绿素和类胡萝卜素含量降低有关, 因酵母双杂及体外免疫共沉淀实验表明VAR3与9-顺式-环氧类胡萝卜素双加氧酶NCED4和NCED5相互作用(Næsted等2004), 而NCED4和NCED5参与植物激素ABA的合成(Huo等2013; Frey等2012)。最近的研究发现, VAR3/OZ1与叶绿体的RNA编辑体中的亚基ORRM1互作, 参与叶绿体RNA的编辑过程(Sun等2015)。因此, VAR3调控叶绿体发育应该与RNA的编辑过程相关。

3 斑叶突变体的抑制子

虽然有不少的斑叶基因被克隆, 但是斑叶产生的分子机理并不清楚。在过去的几年中, 主要通过遗传学手段筛选斑叶突变体的抑制子来研究斑叶形成机理, 斑叶基因包括*VAR2*、*THF1*和*IM*。除了*THF1*缺失突变体的斑叶表型是由于FtsH复合体水平下降所引起的报道外(Zhang等2009), 斑叶基因之间的遗传关系研究不多。

到目前为止, 已经报道的*var2*抑制子有*clpC1*、*clpC2*、*clpR1*、*fug1*、*sco1*、*svr1*、*svr7*、*svr3*、*svr4*、*svr8/prpl24*、*svr10*和*bpg2*等(Qi等2016; Liu等2010a, 2010c, 2013; Yu等2008; Miura等2007; Park和Rodermel 2004), *thf1*抑制子包括*clpR4*、*prpl11*、*sig6*、*sig2*、*ys1*、*noa1*、*bpg2*、*prps9*、*prps5*和*sot1*等(Hu等2015; Ma等2015; Wu等2013, 2016)。抑制*thf1*斑叶表型的基因均能恢复*var2*斑叶表型, 反之亦然(Hu等2015; Ma等2015; Wu等2013, 2016)。同样, 过表达异三元G蛋白GPA1也能恢复*thf1*和*var2*的斑叶表型(Zhang等2009)。*im*斑叶表型抑制子的筛选及基因克隆结果表明: 过表达线粒体和叶绿体双定位的*AOX2*能够恢复*im*的斑叶(Fu等2012)。此外, 参与PSI循环电子传递的CRR2 (chlororespiratory reduction)或者PGR5 (proton gradient regulation 5)的功能缺失突变也能恢复*im*的斑叶表型, 意味着PTOX、CRR2和PGR5共同参与PQ库的调控, 并对叶绿体的早期发育起着重要的作用(Okegawa等2010)。

归纳已报道的*var2*与*thf1*斑叶抑制基因, 我们发现大部分的基因功能与叶绿体基因表达包括转录和翻译过程密切相关。例如, 参与叶绿体基因翻译的基因有*FUG1*, 编码定位于叶绿体的翻译起始因子2 (translation initiation factor 2), 以及*SCO1*和*SVR3*, 它们分别编码定位于叶绿体的翻译延伸因子G和A (translation elongation factor G and TypA-like) (Liu等2010a; Miura等2007)。这与叶绿体翻译抑制剂氯霉素(chloramphenicol)和壮观霉素(spectinomycin)处理以及一些编码质体核糖体蛋白的基因(*prpl11*、*prps9*、*prps5*、*svr8/prpl24*)突变抑制*var2*或者*thf1*斑叶表型的结果相一致(Ma等2015; Liu等2013; Wu等2013)。同样, 控制质体基因表达的一些基因突变也能抑制*var2*和*thf1*的斑叶。例如, SIG6和SIG2的突变能够抑制*thf1*的斑叶表型, 而其它的σ因子则不能(Hu等2015); *YS1* (*Yellow Seedling1*)编码一个PPR蛋白, 其突变导致RNA聚合酶(PEP)亚基RPOB的转录本编辑异常, 使PEP酶活性降低, 从而抑制*thf1*以及*var2*的斑叶表型(Hu等2015)。

核糖体是蛋白质的翻译场所, 其组装受到rRNA加工与修饰、核糖体蛋白的有序结合等生物学过程的调控。*SOT1*编码一个定位于叶绿体的PPR蛋白, SOT1蛋白23S-4.5S rRNA前体的5′端结合, 一方面保护23S-4.5S rRNA前体的5′端免受核

酸酶的降解, 另一方面可以帮助形成rRNA的二级结构促进rRNA的加工和成熟。因此*SOT1*突变导致成熟rRNA产物减少, 从而影响叶绿体蛋白翻译速率降低而抑制斑叶的表型(Wu等2016)。拟南芥中与*SOT1*同源性最高的基因*SVR7*突变也影响叶绿体rRNA的加工, 但其作用机理还不清楚(Liu等2010c)。*sot1*和*svr7*分别能抑制*var2*和*thf1*的斑叶表型(Wu等2016)。另外的抑制子包括*noa1/rif1/svr10*, 其功能与细菌中参与30S核糖体生物发生的YqeH蛋白高度同源(Qi等2016; Flores-Pérez等2008; Loh等2007; Uicker等2007), 以及*bpg2*, 一个结合叶绿体16S和23S rRNA的蛋白(Kim等2012)。

*var2*和*thf1*的抑制子筛选中, 发现多个叶绿体Clp蛋白酶复合体的亚基突变均能回复斑叶表型, 包括ClpC1、ClpC2、ClpR1与ClpR4 (Wu等2013; Yu等2008; Park和Rodermel 2004)。叶绿体蛋白质组分析结果表明*clpR4 thf1*的蛋白质谱与*clpR4*的相关性显著高于*thf1*, 证明了遗传上*clpR4*上位于*thf1*, *clpR4-3 thf1*中斑叶被抑制至少部分是因为FtsH蛋白含量较*thf1*有所增加; 而Clp相关亚基的突变引起质体核糖体不能正常组装, 导致叶绿体发育减慢(Wu等2013)。高等植物Clp蛋白酶复合体虽起源于原核生物, 但其组成成员得到很大扩展(Adam等2006)。Clp蛋白酶是由3层圆形的核心亚复合体(ClpP、ClpR、ClpT)组成, 在识别底物时还需要其他一些成员参与(ClpF、ClpS1) (Nishimura等2015)。近年来, 叶绿体Clp蛋白酶的作用底物的鉴定、识别与降解过程也取得了较好的研究进展(Apitz等2016; Pulido等2016)。

*im*突变体的斑叶抑制子则有所不同。研究表明: 在叶绿体中过量表达AOX2、AOX1a能够抑制*im*的斑叶表型, 同时可以纠正*im*突变体中八氢番茄红素的异常积累。AOX2、AOX1a在叶绿体中过量表达时, 可以替代PTOX行使功能, 从而抑制*im*突变体斑叶的表型(Fu等2012)。循环电子传递损伤的*pgr5*和*crr2-2*突变体也能抑制*im*的斑叶表型(Okegawa等2010)。这些结果显示PQ库的平衡具有调控叶绿体发育的作用。此外, 与控制昼夜节律中心元件GI突变可以抑制*im*突变体生长发育后期的斑叶表型, 推测*gi2*突变带来的增加光合作用以及抗氧化能力, 从而释放了*im*中的还原压力(Putarjunan和Rodermel 2014)。因此, *im*突变体的抑制子与*var2*和*thf1*的抑制子明显不同, 它们主要通过补偿PTOX的功能以及释放PQ库的还原压力来达到抑制斑叶的目的(Foudree等2012)。

4 斑叶形成假说

4.1 阈值模型

叶绿体发育及其功能维持受到众多的生理生化过程的调控。在叶片发育的早期, 叶绿体发育过程陪伴着质体和细胞的分裂, 质体之间存在异质性。因此, 每个质体发育的潜力有可能是不同的。FtsH复合体对叶绿体发育与功能很重要。当FtsH2或者FtsH5缺失的情况下, 有些质体能获得足够多的FtsH各个亚基, 并组装成有功能的FtsH复合体, 而有些则不能获得足够的FtsH (图2)。因此, 叶绿体发育对FtsH复合体的量有一个最低的要求, 即所谓的阈值。当质体的FtsH水平超越阈值时, 类囊体就能形成, 而处于阈值以下的质体则不能正常发育, 停留在未分化阶段。但是, 未发育质体的细胞可以从周围绿色细胞中获得所需要的营养, 进行正常分裂与分化, 从而形成黄/白色区域(Kato等2007; Yu等2007; Aluru等2006)。在阈值假说中, 叶绿体发育所需的阈值不是固定不变的, 它会随着生长环境和生理状态而改变(Miura等2007)。阈值假说获得FtsH同源基因互补以及遗传抑制子筛选等实验结果的支持(Wu等2013; 2016; Kato等2007; Zaltsman等2005a; Yu等2004, 2007)。

斑叶突变体*thf1*和*var2*的斑叶形成就可以由FtsH的阈值假说来解释(图2)。斑叶表型恢复的机制可能通过以下两种途径: 一种是由于核基因的突变直接或者间接提高了FtsH复合体的水平, 最终促进叶绿体发育, 例如过表达GPA1提高了FtsH的基因表达, Clp复合体的突变也提高了FtsH的积累水平; 另一种可能是因核基因突变导致FtsH的阈值下降, 因而提高叶绿体发育的比例。但是我们用蛋白印迹技术检测发现斑叶抑制子中的FtsH包括VAR1和VAR2的水平并没有下降。

4.2 蛋白质合成与降解平衡假说

叶绿体蛋白合成与降解平衡假说是基于研究*var2*抑制子的作用机理上提出的。遗传筛选发现许多叶绿体蛋白质合成相关的基因突变均能恢复*var2*的斑叶表型。在*var2*突变体中, 因为FtsH复合

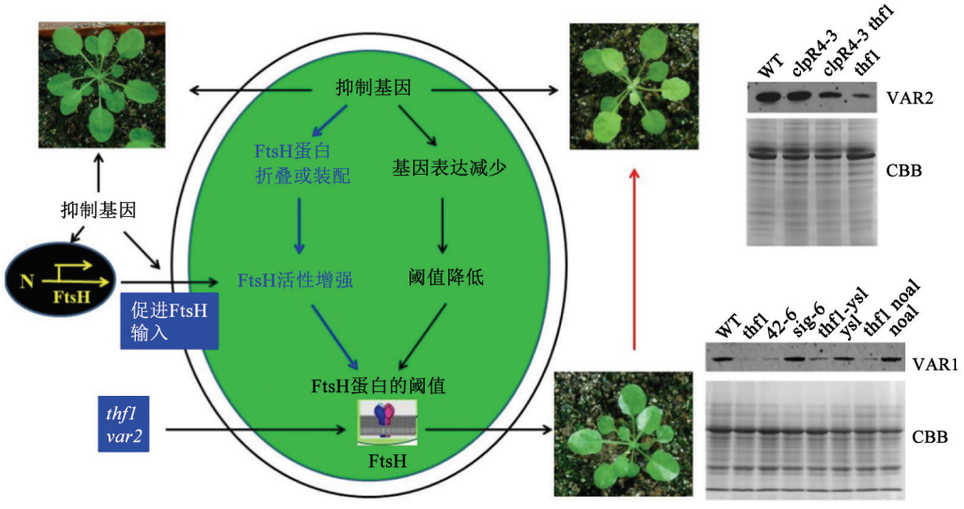

图2 抑制斑叶发生的模型与FtsH在各种突变体中的表达水平

Fig.2 A model for suppression of leaf variegation and FtsH expression levels in various mutants

体水平下降, 叶绿体蛋白不能及时被降解而积累, 抑制叶绿体发育; 叶绿体蛋白合成相关基因突变导致蛋白质翻译速度减慢, 使蛋白合成和降解达到平衡。同时, 叶绿体蛋白质合成受阻, 延长了叶绿体发育时间, 有可能进而降低斑叶形成所需要的FtsH阈值, 促进叶绿体发育(Hu等2015; Ma等2015; Wu等2013, 2016; Liu等2010b, c; Yu等2008; Miura等2007)。因此, 阈值和蛋白质合成与降解平衡这两种假说并不矛盾。

4.3 光氧化假说

光氧化假说的主要证据来自斑叶突变体的叶绿体发育缺陷随着光强增强而变得严重(Rosso等2006), 但是也有报道*var1*和*var2*突变体斑叶的形成不是因为光氧化引起的, FtsH蛋白突变引起的光氧化和斑叶的形成可能是两个相对独立的过程(Kato等2007, 2009; Zaltsman等2005b)。这与斑叶突变体中观察到黑暗条件下某些细胞黄化质体发育受到阻碍的结果是一致的(Wu等2011)。

5 展望

斑叶突变体发现至今已有一个多世纪了, 随着斑叶相关基因的克隆以及功能解析的进展, 我们对斑叶形成有了一定的了解。当基因突变影响到某些代谢途径(Yu等2007; Hung等2004; Næsted等2004; Joët等2002; Aluru等2001, 2006, 2007; Cournac等2000)、细胞器基因组稳定性(Maréchal等2009; Abdelnoor等2003; Sakamoto 2003), 以及质体功能(Wang等2004; Sakamoto等2002, 2003; Chen等1999)等方面时, 最终都影响叶绿体发育, 导致斑叶形成。但是关于斑叶形成的分子机制仍然很清楚。例如, 突变体细胞在质体分化早期如何决定发育成黄/白色区域还是绿色区域, 植物特定发育阶段的生长环境和生理环境如何决定花斑形成(Liu等2010b; Miura等2007; Rosso等2006)。因此在分子水平上阐明斑叶形成的分子机制是今后研究中的最大挑战。

值得一提的是我们的研究发现斑叶突变体之间存在着一定的遗传学关系。*thf1*突变体中FtsH含量显著下降, *thf1 var1*和*thf1 var2*的表型分别与*ftsh1 var1*和*ftsh8 var2*表型一样。因此, *thf1*突变体斑叶的形成是由FtsH含量减少所引起的(Zhang等2009)。比较有趣的是很多逐渐转绿的突变体都能抑制*var2*和*thf1*的斑叶表型, 是否也能抑制其他斑叶突变体有待研究。*var2*和*thf1*抑制子的筛选发现Clp蛋白酶复合体相关亚基ClpR1、ClpR4、ClpC1、ClpC2的突变抑制斑叶表型(Wu等2013; Yu等2008; Park和Rodermel 2004), 说明同属AAA蛋白家族的Clp蛋白酶复合体与FtsH蛋白酶复合体之间存在某种关系。因此, FtsH、Clp和THF1的生化关系值得深入研究。

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A review of leaf variegation mutants and mechanisms in *Arabidopsis*

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Abstract: *Arabidopsis* leaf variegation mutants provide us with ideal materials to study chloroplast development since the degree of leaf variegation depends on genetic and environmental factors, and their interactions. How green/white sectors are formed in the same genetic background is an interesting question that needs to be addressed. To date, more and more evidence has shown that most of genes identified from genetic screening for suppressors of the leaf variegation phenotype are directly involved in various biochemical processes of chloroplast gene expression, deepening our understanding of molecular mechanisms on chloroplast development.

Key words: leaf variegation; chloroplast development; suppressors; chloroplast gene expression; *Arabidopsis*

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